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THE TREATMENT OF INTUSSUSCEPTION

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THE purpose of this paper is to bring forward evidence in favor of the treatment of intussusception by a preliminary injection per rectum, before resorting to operation; since by so doing, one will find that about 60 per cent of all cases will be completely reduced, and by carefully observing certain signs, one can be quite certain of the completeness of the reduction in 40 per cent of all cases coming under treatment. Of the cases which will require open operation, either because of the uncertainty of complete reduction or because of the certainty of nonreduction, at least 18 per cent will be found reduced at operation. Moreover, one will find that in many of the cases operated upon, because of the doubtful result of the injection, a large number will be found at operation to be almost completely reduced, so much so that it is quite obvious a more prolonged attempt at reduction by injection would have proved successful.

The only rational method of reducing an intussusception would appear to be by applying pressure on the apex of the intussusception from within the bowel; even when operating on these cases, it is this method which is actually employed, although it is done manually through the bowel wall, instead of by water pressure from within the bowel. The only reasonable objection to the employment of the latter method is the difficulty in the diagnosis of complete reduction. The danger of rupturing the bowel can be disregarded, if precautions are taken to use a definite pressure, well below that likely to cause damage to the bowel wall; I have found that the pressure of a column of water,

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three feet six inches in height, which is equal to a column of mercury of sixty-five millimeters, is quite safe. Many years ago I had one case where the intussusception had been present for three days, and where a ring of ulceration was present in the ileum at the apex of the intussusception and a pinhole perforation occurred after using hydrostatic pressure. In this case, although a column of water of the height recommended had been used, the water was held in the bowel, while the abdomen was being palpated to determine the extent of the reduction; so actually a very much greater pressure than that recommended was used. Hence I regard palpation of the abdomen during the actual attempt at reduction by hydrostatic pressure as a dangerous procedure.

From a perusal of the literature on the subject of intussusception, it is obvious that most surgeons unhesitatingly condemn any other method of reduction except open operation; and the statements made show that in most cases, the writers have neither made themselves familiar with the very simple technic nor have they realized that there are certain signs by which one can be quite certain that reduction has been effected. The argument most frequently used in favor of open operation in every case is that by so doing one can be certain that reduction has been effected; and another is that in these days, surgery has reached such a state of perfection that an open operation is quite safe and free from risk.

Now any open abdominal operation on a young infant is attended by a certain mortality, even though it may be a low one; and it is quite obvious that any operation involving extensive handling of the bowel, especially in a young infant already greatly shocked by the telescoping of the bowel and poisoned by the resultant toxemia, will be attended by a very considerable mortality; moreover, an open operation is generally followed by intraabdominal adhesions. It has been my experience when operating for a recurrent intussusception that adhesions are almost invariably present as a result of the first operation. The closure of a midline incision in a very young infant without the risk of some adhesions to the peritoneal scar is a very difficult matter. Adhesions following an abdominal operation in an infant, particularly if the small bowel has become adherent to the abdominal wall, are far more serious than similar adhesions in an adult, because of the probable unequal growth of the segments of the bowel on the proximal and distal sides of the adherent loop, thereby increasing the chances of kinking and obstruction. I have seen several cases of acute intestinal obstruction in children of from six to ten years of age due to kinking of the bowel as a result of adhesions following an operation for intussusception in infancy. A brief history of such a case is as follows:

CASE REPORT.—Female, aged ten years, was admitted to the Children's Hospital as a case of appendicitis, and the appendix was removed. The pain and vomiting

became more acute after the operation, signs of intestinal obstruction developed, and the child died. At postmortem the obstruction was found to be due to the adherence of a loop of small bowel to an old scar in the midline, through which an operation had been done in infancy for the reduction of an intussusception.

I can recall another case of a child developing intestinal obstruction when six years of age, and where an operation for intussusception had been performed by the late Sir Charles Clubbe when the child was six months old. In this case, through-and-through silkworm-gut sutures had been used to unite the abdominal wound in a single layer as was the custom at one time, and since such suturing leaves a ladder of silkworm gut on the peritoneal surface of the incision, adhesions frequently of a dangerous nature must necessarily result.

Apart from these remote risks there is always the danger of obstruction or peritonitis being an immediate result of an open operation. One of the deaths in my own series was due to general peritonitis following an open operation; in this case, the intussusception had been reduced by a preliminary injection. Another case illustrating the danger of open operation was one in which I had successfully reduced an intussusception of over eighteen hours' duration in a young infant. The intussusception recurred six months later, and although early identification of the nature of the trouble was made by the mother who recognized the symptoms as being similar to those present in the first instance, the child being brought under treatment within three hours of the onset and operated upon immediately by a surgeon who had successfully operated on numerous cases of this kind, the child died of general peritonitis seven days later.

These are not isolated cases and are mentioned merely to emphasize the fact that operations on young infants are not free from some risk; even though they may recover from the operation, there is still a risk of trouble developing later on, owing to the presence of adhesions within the abdominal cavity.

Of course, it is not possible to avoid operation in every case, but where such interference can be avoided, both the immediate and late mortality rates, and the morbidity rate will be lowered.

As to the question of the reliability of the method of reduction by hydrostatic pressure, I might mention that after using this method as a routine for over twenty years, in no single instance has a mistake been made in sending a patient back to bed from the operating theater incompletely reduced, where a diagnosis of complete reduction has been made.

TECHNIC OF METHOD OF HYDROSTATIC PRESSURE

Normal saline solution is used for the injection. The quantity varies with the age of the child, but it is the pressure used, not the quantity of solution, which is the important factor. The pressure of a column

of saline solution three feet six inches high is used. I believe the bowel is liable to rupture from a pressure of five feet, so that there is a good margin of safety. A general anesthetic is administered, and the treatment is carried out in a room adjoining the operating theater and is not done on the operating table. Both patient and surgeon are prepared as for an open operation; so that beyond changing gloves, no time is lost if an operation proves to be necessary. An ordinary douche may be used, but a glass container where the level of the water can be seen is better. The vessel is suspended so that the height of the column of saline solution is three feet six inches above the table on which the child is lying. A No. 15 soft rubber catheter is inserted a few inches into the rectum, and the buttocks are pinched together

Fig. 1.—Illustrates the method of retaining catheter in rectum during injection under pressure.

around it so as to prevent any saline solution from escaping (Fig. 1). No lubricant is used, as otherwise it is difficult to hold the catheter in the bowel. As the saline solution slowly runs in, one can follow the outline of the distended colon. After about three minutes the catheter is disconnected and the saline solution allowed to escape into a bed pan. The return of this first injection generally clears out most of the blood and mucus and possibly some feces from below the obstruction. The process is repeated twice, giving about three minutes on each occasion. Occasionally I have used a single injection, holding it in the bowel for from eight to ten minutes. After the injection, as much of the saline solution as will come away is allowed to escape, so that the large bowel is left practically empty after the treatment. On several occasions I have used a thin barium solution instead of saline

solution; on such occasions an x-ray film has been taken to demonstrate the presence of the opaque fluid in the small bowel (Fig. 2).

SIGNS OF COMPLETE REDUCTION

1. *Abdominal distention.*—There are several signs indicating probable reduction, but the only trustworthy sign indicating complete reduction is abdominal distention after the injection which remains

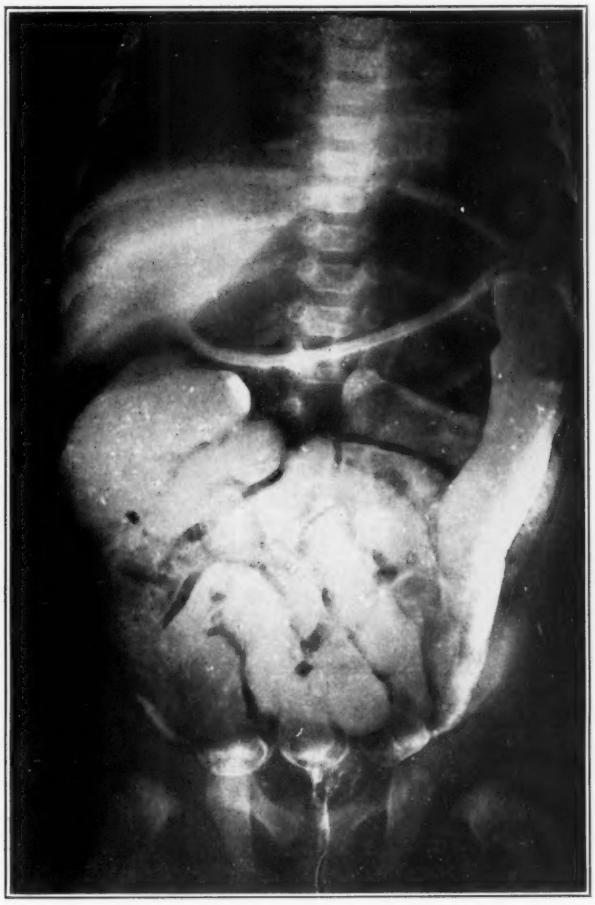

Fig. 2.—An x-ray after thin barium injection retained under pressure for ten minutes. Note opaque solution in coils of small bowel.

after the saline solution has been allowed to escape from the large bowel. The distention is quite obvious on inspection and feels firm on palpation. One is obviously palpating an abdomen where a large portion of the small bowel is distended by fluid.

In most cases of intussusception of under twenty-four hours' duration, the abdomen is soft and flaccid when first seen, although in long-standing cases the abdominal distention of intestinal obstruction is present; in such cases, an operation should always follow the injection

which can be used, however, as a preliminary measure. In early cases, therefore, the intussusception can always be felt by abdominal palpation. After complete reduction following injection, provided the saline solution has passed into the small bowel, it would be impossible to palpate a tumor, even if one was present in the abdominal cavity, and so under such circumstances one can be quite certain that the intussusception has been reduced. When, however, it is possible to palpate a mass in the abdomen (usually in the ileocecal region), such a mass may be due either to thickened and edematous bowel following reduction or to an unreduced intussusception, and an operation will be necessary to determine which condition is present. Such an operation can usually be done through a muscle-splitting incision, similar to that

Fig. 3.—Illustrates the flaccidity of abdominal wall, after injection under pressure, where none of the injection had gone through the ileocecal valve. Such a case may or may not be reduced, but in either event, a mass remains palpable.

commonly used for the removal of an appendix. (See Fig. 3 where a mass could still be palpated after injection.)

2. *Alteration in Circumference of Abdomen.*—Measurement of the circumference of the abdomen at the level of the umbilicus before and after treatment will usually show an increase of about two inches where the intussusception has been reduced.

3. *Yellow Fecal Material in the Returned Saline Solution.*—All fecal material below the obstruction is usually blood-stained. If the second or third return contains yellow particles of feces, these have usually come from the small bowel above the obstruction.

4. *Flatus in the Return.*—Provided one is careful not to allow any air to pass into the bowel with the enema, the presence of flatus after the first injection is evidence in favor of complete reduction.

5. *X-Ray Film After Opaque Injection.*—When thin barium solution has been used instead of saline solution, the presence of some of this in the small bowel, as seen by x-ray examination, would indicate complete reduction.

6. *Use of Charcoal to Verify Reduction.*—On a number of occasions I have left about one teaspoonful of powdered charcoal in the stomach by suspending it in about one-half ounce of water and passing a stomach tube. In cases of complete reduction, some of the charcoal, which is very easy to identify, can be recovered within five hours by washing out the large bowel. Although I do not regard this sign as one to be used to decide the question of open operation, still where the intussusception is of only a few hours' duration and where reduction by injection has been attempted, one can obtain by this means absolute confirmatory evidence of complete reduction, even in the absence of abdominal distention and even in cases where there is other positive evidence of complete reduction; the giving of charcoal in this way gives an added feeling of security, because if one could not recover

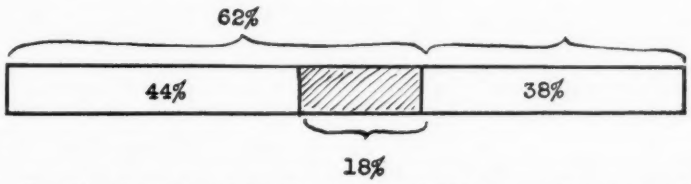

Fig. 4.—Results of treatment by the author, of 100 consecutive cases of intussusception by hydrostatic pressure. 62 per cent reduced by hydrostatic pressure; 44 per cent did not require operation; 18 per cent reduced by injection, but small incision made to verify reduction; 38 per cent required operation because intussusception not completely reduced.

some of the charcoal within six hours by washing out the bowel, it would be wise to consider the possibility of incomplete reduction.

7. *Opaque Enema as a Means of Diagnosing Reduction.*—An opaque enema can be used, and by fluoroscopy, the actual reduction of the intussusception can be followed; but as the long and mobile sigmoid and transverse colon are distended with the opaque fluid, it is difficult to see the cecum until most of the enema has been allowed to escape. This method, therefore, is not a practical one and, in my opinion, has no advantages over the simple saline injection; moreover, more time is wasted if one has to proceed to operation.

In a series of 100 cases the duration of the different groups was as follows:

(1) Reduced by hydrostatic pressure alone.
Average duration 16 hours. There were ten over 24 hours; three over 48 hours; and one over three days.

(2) Reduced by hydrostatic pressure followed by operation.
Average duration 17 hours. There were two over three days.

(3) Cases operated upon because of failure of injection.
Average duration 26 hours.

SUMMARY OF RESULTS OF TREATMENT BY THE AUTHOR IN 142 CONSECUTIVE CASES OF INTUSSUSCEPTION; 92 MALES AND 50 FEMALES

1. Sixty cases cured by hydrostatic pressure alone, no operation done:

	No. of cases	
Ages 1 to 3 months	1	
3 to 6 months	16	
6 to 9 months	23	
9 to 12 months	12	
12 to 18 months	5	
Over 2 years	3	
	60	All recovered.

2. Thirty cases treated by hydrostatic pressure, followed by operation to verify reduction:

	No. of cases	
Ages 1 to 3 months	1	
3 to 6 months	10	
6 to 9 months	9	
9 to 12 months	6	
12 to 18 months	2	
Over two years	2	
	30	Recovered, 29
		Died, 1

3. Operation performed because of inability to accomplish reduction by hydrostatic pressure:

	No. of cases	
Ages 3 to 6 months	10	
6 to 9 months	15	
9 to 12 months	17	
12 to 18 months	4	
Over two years	6	
	52	Recovered, 46
		Died, 6

Total deaths in 142 cases, 7. Mortality, 4.9%.

Since space will not permit of a description of all the cases treated, certain of them have been selected either as being typical or as demonstrating certain features which are of importance in carrying out the treatment outlined.

CASE 1.—D. H., female, aged nine months. Duration of symptoms forty-one hours. A very healthy looking child. Large mass palpable in left side of abdomen. Three injections were given, but a small mass could still be felt in the right iliac region. A muscle-splitting incision was made in the right iliac region, and the cecum ascending and transverse colon were congested, evidently having been involved in the intussusception. The ileal portion of the intussusception remained, and was reduced with difficulty. It had started nine inches from the ileocecal valve. The child recovered.

CASE 2.—V. B., male, aged eighteen months. Had been ill with repeated attacks of screaming and pallor for three and one-half days. Had passed a very little blood-stained mucus but no feces. Had been vomiting at intervals. A mass about eight

inches long could be felt about the umbilical region. Three injections were given; the first returned almost clear; the second contained a good deal of blood. After the third, a mass about two inches long was still palpable. A right muscle-splitting incision was made, and a small unreduced intussusception was present in the ileum. This was reduced with difficulty, and was found to commence about eleven inches from the ileocecal valve. The child made an uninterrupted recovery.

Fig. 5.—Abdomen distended after injection, but since palpation still revealed a mass due to a small unreduced portion of intussusception, it was obvious that the distention was not due to injection having passed into small bowel.

Fig. 6.—Same patient as shown in Fig. 5. The last portion of an ileocolic intussusception being reduced by gentle traction on ileum.

Comment.—This child was under the care of a pediatrician who had treated many cases of intussusception. He saw the case twelve hours after the commencement of the trouble but was misled because of the

almost complete absence of blood from the rectum. It was only after a portion of the intussusception had been reduced by the first injection that any quantity of blood appeared.

CASE 3.—K. D., male, aged five months. Had been in hospital for seven days under treatment as a case of gastroenteritis, although nothing but blood and mucus had been passed during this period. On the seventh day a tumor could be felt both through the abdominal wall and per rectum; the case was diagnosed as intussusception. The infant was extremely emaciated and was obviously too ill for any operative interference. Three other surgeons who saw this case agreed that operation was out of the question. The mass disappeared after a third injection, and there was no further sign of gastroenteritis, indicating that the case was one of intussusception from the first. The child made an uninterrupted recovery.

CASE 4.—E. B., female, aged six months. Duration of intussusception, four hours. Mass which was palpable before treatment, disappeared after injection and abdomen remained distended. One teaspoonful of charcoal was left in the stomach, and some of this was recovered by rectal lavage five hours later.

CASE 5.—V. K., female, aged seven months. Duration of intussusception, ten hours. Abdomen distended after third injection. One teaspoonful of charcoal left in stomach after treatment. Some of this recovered by rectal lavage six hours later.

CASE 6.—R. M., male, aged five years. Sudden onset with abdominal pain and signs of shock five hours before admission to hospital. Was sent to hospital as case of appendicitis. A freely movable mass could be felt in the right iliac region, and some blood was present in the rectum. The mass disappeared after injections of saline solution, but abdomen remained flaccid. Abdomen was opened through a right-sided muscle-splitting incision. The cecum and last few inches of ileum were greatly thickened, but the intussusception had been reduced.

CASE 7.—C. D., male, aged four months. Duration of intussusception, twenty-four hours. Was first admitted to medical ward, as no blood was seen after enema. Later a mass was palpable in region of hepatic flexure of the colon. This disappeared after injection, but as abdomen did not remain distended, an operation was done. The intussusception had been reduced by the injection; the cecum and ascending colon showed evidence of having been involved in an intussusception.

CASE 8.—G. H., male, aged nine months. Duration of symptoms, twelve hours. Mass palpable in ileocecal region. Three injections given, but abdomen did not remain distended, hence operation was done. A very small portion of the intussusception was found unreduced and was reduced with such ease at operation that it was quite obvious that if one had persisted a little longer with the injection, it would certainly have yielded to hydrostatic pressure.

CASE 9.—D. H., male, aged eight months. Duration of symptoms seventy-two hours. Intussusception palpable per rectum. The patient had been treated as a case of gastroenteritis and had been given a considerable amount of a bismuth preparation. After the second injection bismuth stained feces came away although nothing but blood and mucus were seen after the first injection.

Comment.—An operation was not done on this case, because the appearance of the bismuth after the injection was an indication of complete reduction. The course of events in this case suggests that if for

any reason operation has to be postponed for say five or six hours, the administration of bismuth or charcoal before attempted reduction by hydrostatic pressure might be of value in diagnosing complete reduction.

BRIEF HISTORY OF THE SEVEN CASES THAT DIED

CASE 1.—Female, aged ten months. Duration of intussusception, five hours. Three injections were given from a height of three feet six inches, followed by operation through lateral gridiron incision. The intussusception was very easily reduced. On the sixth day an abscess developed in the right iliac fossa, and the wound broke down. The pus was evacuated and a tube was introduced, but the child died of general peritonitis thirteen days after operation.

Comment.—This child died as a direct result of infection following operation. The ease with which the intussusception yielded at operation suggested that a more prolonged attempt at reduction by hydrostatic pressure would have been successful in reducing the intussusception, thus avoiding the fatal operation.

CASE 2.—Male child, aged three months. Duration of intussusception, three days. Very collapsed on admission to hospital. One injection was given and was followed by immediate operation. Intussusception was quite irreducible, and about eighteen inches of bowel were resected, followed by lateral anastomosis. Child died four hours later.

CASE 3.—Female, aged fifteen months. Patient had been under treatment for five days as a case of gastroenteritis, although from the history it was obvious that it was an intussusception from the start. On the fifth day, about five inches of the bowel prolapsed through the anus, and the real nature of the trouble was diagnosed. The prolapsed portion of the intussusception was returned through the anus, and three injections were given. These considerably reduced the size of the mass palpable in the abdomen. Operation followed through a midline incision. The whole of the large bowel was greatly thickened and edematous, and the remaining portion of the intussusception was easily reduced. The whole of the large bowel was extremely mobile, the case being one of nonfixation of the colon. It was the mobility of the colon which had prevented dangerous strangulation of the bowel and enabled reduction to be effected with such ease. The child died from shock six hours after operation.

Comment.—If this child had been given plenty of saline solution before operation, or if operation had been preceded by a transfusion of blood and if a more prolonged attempt at reduction by hydrostatic pressure had been made before resorting to operation, I believe the intussusception would have yielded without operation, and the child's life would have been saved.

CASE 4.—Female, aged ten months. Duration of intussusception, three days. One injection given, followed by immediate operation. Intussusception found to be quite irreducible. About twelve inches of bowel were resected, and Paul's tubes were inserted into the open ends. Death followed eight hours later. Examination of resected portion showed that the intussusception had commenced in the ileum about eight inches from the ileocecal sphincter.

CASE 5.—Female, aged eight months. Duration of intussusception, four days. Child had been treated as a case of gastroenteritis for four days, although the history showed that this diagnosis was incorrect. The child was admitted to hospital in an extremely collapsed condition. Three injections were given, and the mass which was palpable before this treatment almost disappeared. Operation followed and the small remaining portion of the intussusception was easily reduced. It was of the ileocecal variety. The child seemed to be doing well but collapsed suddenly and died twenty-four hours after operation.

CASE 6.—Male child, aged nine months. Duration of intussusception, fifty hours. On admission to hospital, he was in an extremely collapsed condition with rapid pulse and sunken eyes. One injection was given and operation followed immediately. The intussusception was irreducible and resection was done. Death occurred three hours later. An examination of the resected segment showed that the intussusception had commenced in the ileum nine inches from the ileocecal sphincter.

CASE 7.—Female, aged ten months. Duration of intussusception, three days. Large mass palpable per rectum, and through abdominal wall. Injection given from height of three feet six inches. Abdomen became distended after injection, but operation was done on account of long history. On opening the abdomen, it was discovered that the intussusception had been completely reduced, and there were indications that it had commenced about three inches from the ileocecal valve. There was a small pinhole perforation in the ileum about three inches from the ileocecal valve, and some of the saline had escaped into the peritoneal cavity. The child died about twelve hours after operation.

Comment.—At postmortem it was found that the perforation was situated on the course of a ring ulcer of the ileum. The ulcer was evidently at the level of what had been the apex of the intussusception. In this case, my assistant had held the catheter in the bowel while I palpated the abdomen before the saline solution had drained away from the large bowel. Hence, a much greater pressure had been used than that recommended, and in addition, the bowel had been weakened by the ulceration due to the long duration of the intussusception. This case occurred as far back as the year 1923 and is the only one of the kind which I have encountered.

A BRIEF REVIEW OF THE SEVEN CASES THAT DIED

Of these seven cases that died, the duration of the intussusception in five of them was three days and over; in one, it was over two days; and in the remaining case, it was only five hours. This case was the only one in the whole series of 142 cases in which death occurred, where the duration of the intussusception was under thirty-six hours.

In three of the cases, resection was done and all died. Case 1 died of general peritonitis following infection as a direct result of the operation. Two children died from shock following operation. One child died as a result of perforation of the bowel.

A study of these cases shows, as is well known, that the most important factor in reducing the mortality rate is early diagnosis and

treatment. Where, however, an intussusception has been present for three or four days, it is far more important to apply measures to counteract the shock and to restore the loss of fluid and chlorides to the blood and tissues than to do an immediate operation.

Where reduction of the intussusception cannot be effected at operation, resection in an infant is attended by a very high mortality, but may be successful if saline injections or transfusion of blood are given beforehand.

Instead of resecting the unreduced portion of bowel, successful results have been reported by doing a lateral anastomosis and then leaving the unreduced portion of the intussusception in situ. Possibly such a procedure might have been successful in one or other of the above cases.

SUMMARY

The treatment of intussusception by a preliminary injection of saline per rectum is advocated, and evidence is adduced in favor of this procedure. Certain signs indicating complete and probable reduction are given. Then follows a brief summary of the results of such treatment of a consecutive series of 142 of the author's cases and a short history of a few selected cases.

THE THERAPEUTIC MANAGEMENT OF INTESTINAL OBSTRUCTION

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THERE is no substitute for early operation when the intestine is mechanically obstructed. All other treatment is adjunct treatment to aid in the conservation of life until the obstruction and its effect upon the patient can be relieved.

Any consideration of the therapy of intestinal obstruction would be incomplete without emphasizing the fact that there are two definite types of obstruction, simple mechanical occlusion of the bowel and occlusion with disturbed blood supply or strangulation. The latter includes obstructions incident to mesenteric thrombosis and intussusception. Disturbed blood supply adds to the immediate danger of obstruction, demanding greater vigilance and more prompt treatment. In planning treatment for obstruction of the intestine, it is important to remember that the nearer the obstruction is to the pylorus, the more rapidly symptoms will develop and the more promptly death will ensue if the obstruction is not relieved. Obstructions in the colon are much less serious than those in the small intestine and present a very different problem in treatment.

The following factors in the treatment of obstructive lesions of the intestine are worthy of separate considerations: (1) Relief of the mechanical obstruction by operation, (2) maintenance of water balance, chemical balance, and nutrition, (3) prevention and relief of distention of the bowel, (4) application of heat to the abdomen, (5) oxygen therapy, (6) bed posture, (7) transfusions.

1. *Relief of the Mechanical Obstruction by Operation.*—The time and method of operative procedure must depend upon the type of obstruction and the condition of the patient. If the obstruction is of short duration and the patient has not been seriously depleted, operation may be done at once. Other patients suffering from dehydration, hypochloremia, abdominal distention, and rapid pulse should always receive preliminary treatment with water, sodium chloride, and gastric suction before operation. While this preliminary treatment is necessary to lessen the operative risk, it is equally true that such treatment should not be prolonged beyond a few hours before surgical relief is instituted. At this point it is appropriate to warn against delaying operation in those patients who show marked general improvement as a result of supportive treatment.

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Simple occlusion of the intestine can usually be relieved by directly removing or releasing the cause of the obstruction. However, certain conditions may be found or complications may arise during the progress of the operation which make it necessary to resect or repair portions of the bowel, thereby adding to the operative hazard. Stripping of the bowel to remove its contents is of doubtful value as shown both clinically and experimentally by Ochsner and Storck.[1] When emptying the bowel is desirable to reduce distention, stripping adds to the danger of infection and shock; and when the bowel is not overdistended and peristalsis is still active, stripping is not necessary.

When strangulation of the gut is suspected, early operation is imperative to avoid the toxic effect of necrosis and the danger of peritonitis as a result of rupture. Release of the constriction interfering with the circulation may be sufficient providing the surgeon is positive that the blood supply returns to the gut, insuring its viability. If irreparable necrosis has resulted, the extent of operation must depend upon the patient's general condition. Frequently it is much safer to exteriorize the necrotic segment and remove it after closing the abdominal wall, leaving the open ends of the gut protruding to be closed at a later date by anastomosis or by the Mickulicz technic. Operative procedures upon acutely distended gut are always dangerous. In a patient acutely ill with obstruction, resection and anastomosis not only consume valuable time, but the danger of peritonitis is great. Leakage from the distended gut of intestinal obstruction usually means disaster.

Enterostomy as a treatment for mechanical intestinal obstruction is always an operation of necessity and not of choice. There are indications for its uses both as a primary and as a supplementary procedure. In those patients who are too ill for exploration and release of an obstruction, enterostomy may be life-saving. After relief of an obstruction, enterostomy is often advisable to drain a markedly distended bowel or to protect a damaged gut wall or suture line. It is not free from danger. Peritonitis and abdominal wall infection may result from leakage of intestinal contents when a tube and sutures are placed in an overdistended gut wall. The danger of enterostomy may be reduced to a minimum by isolating a segment of bowel with intestinal clamps and by aspirating all gas and liquid content before placing sutures and introducing the tube. Thoughtful care must be used in selecting the proper patients for enterostomy. An error leading to a fatality may be made if enterostomy alone is done for an obstruction due to a strangulated section of the bowel. When an abdomen is opened and bloody fluid is found, the surgeon should definitely assure himself that no strangulation of the gut is present before enterostomy is done. Enterostomy will not successfully drain the bowel if it is

paralyzed. Peristalsis must be present to force the liquid and gas content of the gut through the enterostomy tube. High jejunostomy is of very doubtful value in the treatment of any type of obstruction. The logical place for intestinal drainage by enterostomy is just proximal to the point of obstruction. It is the author's opinion that the suction method emphasized by Wangensteen[2] is far more efficient in emptying the stomach and upper intestine than high jejunostomy.

When an enterostomy is done, the technic of Witzel or one of its modifications,[2] using a size 16 or 18 French rubber catheter, is the safest operation. After the catheter is fixed in the gut, it should be passed through the omentum and when possible the omentum fixed to the suture line for added protection against infection and adhesions.

2. *Restoration and Maintenance of Water Balance, Chemical Balance, and Nutrition.*—In any obstruction of the small intestine, dehydration soon becomes a factor of prime importance due to lack of liquid intake and vomiting. To restore and maintain a sufficient supply of water for the proper functioning of the chemical processes of the body is one of the most essential phases of the treatment of bowel obstruction. As emphasized above, the parenteral administration of water and sodium chloride should precede operation in all cases showing the effects of dehydration and should be continued after operation until the patient is able to drink and retain a sufficient quantity of water to supply the body needs.

The initial quantity of water needed varies much, depending upon the anatomic location and duration of the obstruction. The general appearance of the patient is a helpful guide. The daily intake for the normal individual under usual conditions varies from two to three liters. No accurate estimate can be made of the actual need of all cases except as they are studied individually. It must be recognized that the lack of normal intake does not represent the total loss of liquid to the body. The secretions into the stomach and upper intestine are also lost by vomiting or through the suction tube. This loss may at first amount to several liters per day (Fig. 1).

By clinical experiments, Maddock and Coller[3] have been able to estimate with considerable accuracy the fluid needs of the dehydrated patient. When a patient is first seen with signs of dehydration, such as a hot dry skin, a dry tongue, sunken eyes, rapid pulse, a slight fever, and insufficient urine output, these authors have estimated that there has been a loss of liquid approximately equal to 6 per cent of the body weight. A patient weighing sixty kilograms would therefore require 6 per cent of 60,000 gm. or 3,600 c.c. as an initial supply of water to relieve existing dehydration. The quantity of water estimated for the daily uses of the adult body are two liters for water

of vaporization (loss through skin and lungs) and one and one-half liters for urine. It is, therefore, evident that a seriously dehydrated patient would require in the first twenty-four hours 3,600 c.c. plus 2,000 c.c. plus 1,500 c.c. to restore and maintain water balance. In addition to this requirement, any losses by vomiting, bleeding, fistulas, diarrhea, or massive exudation should be estimated and the quantity restored to the body. After the initial supply of water, the average daily, necessary intake for adults may be estimated as 3,500 c.c. This quantity should be decreased as the patient is able to retain food and liquid given by mouth.

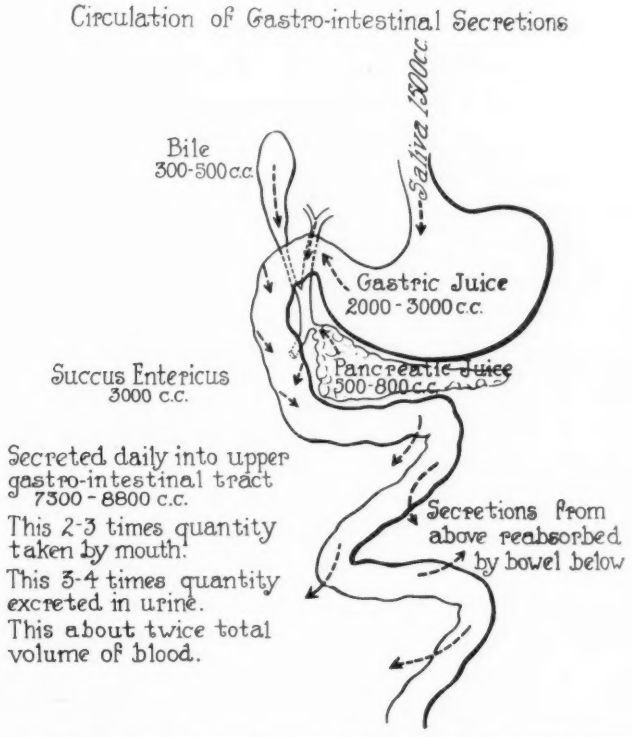

Fig. 1.—Sketch illustrating a circulation of the alimentary tract secretions. This shows the possibility of great fluid loss with obstructions high in the small intestine.

Obstructions of the intestine causing a loss of upper alimentary tract secretions are often associated with marked chemical changes in the body which may be demonstrated by blood examination. This loss is particularly marked if the obstruction is high or prolonged. The important blood changes are a loss of chlorides (hypochloremia) and increases in carbon dioxide combining power and an increase in the nonprotein nitrogen. After prolonged starvation, a disturbance in nutrition may be sufficient to decrease the blood serum protein.[4, 5] Chemical changes assume less importance in those cases in which there

is early relief of the obstruction and in colon obstructions. The best gauge of the extent of body chemical change is an estimation of the blood chlorides. In the treatment of intestinal obstruction, the blood chlorides should be restored to normal and maintained within normal limits. By so doing, the acid-base imbalance is corrected, destruction of body protein minimized, and proper water distribution to the body tissues assured. After a marked reduction has occurred in the blood serum protein, excessive quantities of sodium chloride may result in edema of the subcutaneous tissues or edema of the lungs.

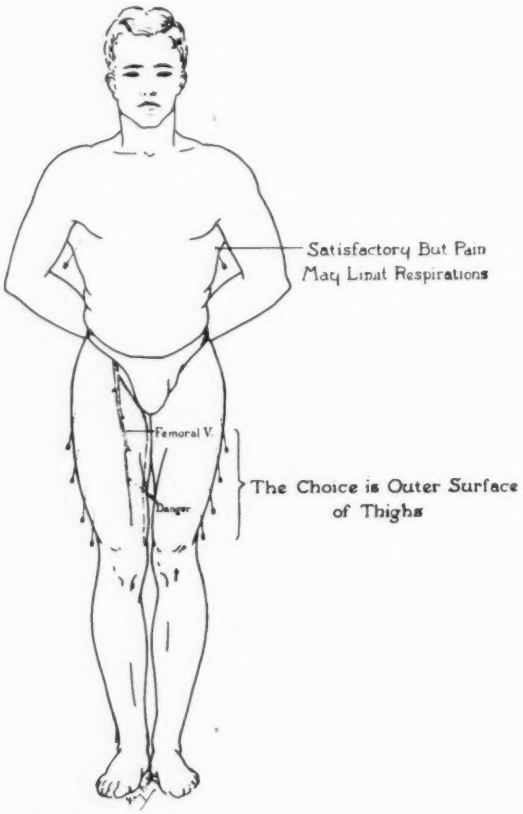

Fig. 2.—Sketch showing choice of location for giving hypodermoclysis.

It is quite evident that metabolic equilibrium cannot be maintained when food cannot be taken by mouth and utilized by the body in a normal manner. It is also quite true that food supply is of much less importance than water supply in the treatment of intestinal obstruction. However, dextrose may be freely given to such patients and will partially furnish the needed nutrition.

Water, sodium chloride, and dextrose may be given in the vein (phleboclysis), under the skin (hypodermoclysis) (Fig. 2), and by rectum (proctoclysis). Water alone should never be given in the vein.

Water is most safely given as physiologic solutions of sodium chloride or dextrose. Patients entering the hospital with marked hypochloremia may be given 250 to 500 c.c. of 2½ or 5 per cent sodium chloride solution by vein to promptly restore the chlorides to normal. In most cases a physiologic solution will suffice. Dextrose in 5 per cent solution is preferable to higher percentages to avoid the dehydration effects of hypertonic solutions. It is unwise as well as unnecessary to give hypertonic solutions under the skin. Subcutaneous tissues tolerate well physiologic sodium chloride solution alone or in combination with 5 per cent dextrose. Ringer's or Hartman's solutions may be used instead of the plain sodium chloride solution and are preferred by many clinicians. Of the methods cited above for administering solutions, phleboclysis and hypodermoclysis are, from the standpoint of quantitative accuracy, more dependable than proctoclysis. Proctoclysis is satisfactory when it works properly, but too frequently the solution is expelled by the sick patient and an accurate estimation of the body intake is impossible. The discomfort incident to increased distention by water introduced into the rectum is also a valid reason for not using proctoclysis. It is also doubtful if dextrose is absorbed by the large bowel in sufficient quantity to be of value.[6]

Solutions injected by any of the three methods under discussion should be given slowly. Fluids given by vein may be safely administered at the rate of fifty to sixty drops per minute. The rate of absorption under the skin varies widely with different patients and should be controlled by preventing tumefaction of the part. A rapid instillation into the rectum will stimulate expulsion and loss of fluid. A very satisfactory routine method of supplying the estimated daily requirement of 3,500 c.c. of fluid is accomplished by injecting 2,000 c.c. into the vein and 1,500 c.c. under the skin. To distribute the intake throughout the day, one of these injections may be given in the forenoon and the other in the evening. The injections should be discontinued at night to avoid disturbing the patient's rest. Since embolism is considered by many a dangerous complication of continuous phleboclysis,[7, 8] intravenous injections in interrupted doses are preferred. To control the salt intake the blood chlorides should be estimated every second day. If the blood chlorides are found normal or above normal, dextrose solution alone should be given until the need of sodium chloride is evident.

There is some danger of giving too much water and sodium chloride. The overburdening of a weakened circulatory system by increasing the blood volume[9] or the production of a general edema and edema of the lungs[10] must be avoided. Too much sodium chloride predisposes to general and pulmonary edema.

3. *Prevention and Relief of Bowel Distention.*—A proper appreciation of the danger of overdistention of the bowel is essential for logical treatment. A patient is usually not dangerously ill unless there is marked distention of the stomach or intestine. Increased pressure within the lumen of the gut seriously interferes with the blood supply of its wall.[11-13] It is probable that there is no absorption of toxic products from the contents of the obstructed intestine until overdistention has damaged the circulation of the gut wall.

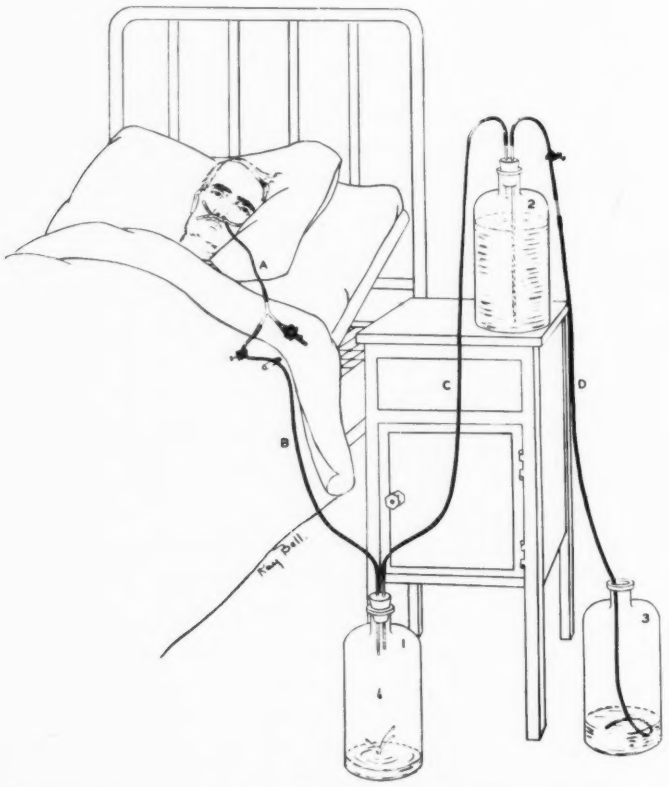

Fig. 3.—Suction apparatus, a modification of suction apparatus described by Wangensteen. *A*, Levin tube and Y-tube attached for ready cleansing with syringe; *B*, rubber tube leading from patient to waste bottle, *1*; *C*, rubber tube connecting waste bottle with water supply bottle, *2*; *D*, rubber tube connecting water supply with overflow water bottle, *3*. Bottles *2* and *3* are interchangeable to avoid new supply of water when *2* runs low. Tube *D* is kept filled with water with lower end immersed in water of bottle *3* to maintain continuous suction.

To relieve distention in the stomach and upper small intestine, continuous suction drainage by an indwelling nasal Levine tube is indicated. Siphonage with suction is superior to simple siphonage, since the former will more readily and completely evacuate the gases as well as the liquid content. The apparatus designed by Wangensteen[2] or one of its slight modifications (Fig. 3) is ideal for this purpose. This method not only reduces the distention but permits the patient to drink water freely which adds greatly to his comfort. The nasal tube

generally causes very little discomfort and may be left in place for several days. The indwelling tube is also of value in testing the return of bowel function. It may be clamped at two- or three-hour intervals when liquid diet is started to determine the tolerance of the bowel for liquid and food. If the aspirated quantity at the end of the closed tube period is less than the intake for that period, the tube may usually be safely removed.

Attempts to reduce distention by repeated enemas are of doubtful value. Enemas should be used with a full realization that gas and feces obtained from the colon by such measures may in no way affect the obstructed bowel above. One must beware of a feeling of false security which might arise after a successful evacuation of the lower bowel. Experiments have shown that the type of enema commonly used does not stimulate peristalsis in the small intestine and cannot be expected to aid in its evacuation.[14]

Enterostomy as discussed above has its value in the mechanical reduction of intestinal distention.

By preventing and reducing distention of the gut, muscle tone and rhythmic contractions are maintained. As long as a bowel retains its power to contract, the blood supply to its walls is intact. A bowel wall cannot be active without an adequate circulation. Since morphine stimulates the tone and rhythmic contractions of the small intestine,[15, 16] it may be given with assurance in sufficient quantity to make the patient comfortable. Since sodium chloride in hypertonic solutions will stimulate peristalsis, it is reasonable to assume that it may be a factor in maintaining bowel tone when given in physiologic solution in sufficient quantity to maintain the chlorides at a normal level in the body.

The use of spinal anesthesia to evacuate the bowel is a rather drastic treatment and of doubtful value. It certainly should never be used for this purpose as long as the bowel is obstructed. Pituitary extract and similar peristaltic stimulants should be used with caution and like spinal anesthesia should not be used as long as obstruction exists. If peristaltic stimulants are used after the obstruction has been released, it is advisable to begin with small doses and increase the dosage if well tolerated. It is the opinion of Ochsner[17] that stimulating drugs are of little or no value in the treatment of ileus.

4. *Application of Heat to the Abdomen.*—Moist or dry heat applied to the distended abdomen is beneficial when judged by clinical observation. It is believed by some authors that heat has a direct effect upon the gut by increasing tone and stimulating peristalsis.[17] Since no known harm results from applying heat, if blistering is avoided, its use is recommended. Heat applied either as moist packs or as dry

heat from electric bulbs over the abdomen seems to be equally effective and may be used as the surgeon's choice dictates.

5. *Oxygen Therapy.*—Oxygen is probably too little used in serious cases of intestinal obstruction. With abdominal distention and changes in blood composition, the vital capacity of the lungs and oxygen carrying capacity of the blood are decreased. The recent work of Fine and his associates[18] presents a new viewpoint in oxygen therapy that may prove to be of value in reducing abdominal distention. They found that the absorption rate of gas within the bowel is increased in direct proportion to the quantity of oxygen given. Thalhimer[19] emphasizes the teaching of Haldane that mild degrees of anoxemia have serious effects upon the nervous system and moderate or severe degrees may be fatal. Oxygen therapy administered by tent, nasal tube, or oxygen room is recommended for patients who are very ill with intestinal obstruction. The administration of oxygen should not be postponed until the patient is cyanotic or the condition seems hopeless.

6. *Bed Posture.*—Comfort and vital capacity are increased by raising a patient's back rest to a semisitting posture. This position may be altered at intervals to rest the patient. Deep breathing and frequent turning of the patient are important to minimize the danger of congestion at the lung bases.

7. *Transfusions.*—The transfusion of blood as a supportive treatment for patients with bowel obstruction has been favorably recommended. It is especially indicated after an illness of several days when protein depletion is imminent.

SUMMARY

It is emphasized again in conclusion that there is no substitute for early operation when the intestine is mechanically obstructed. Inflammatory obstructions due to paralysis or recent adhesions between loops of bowel may spontaneously release themselves with control of distention and other supportive treatment.

Extensive operations upon an acutely obstructed bowel are hazardous except in the early cases. The briefest operation with the least possible trauma is the procedure of choice. It is in this type of obstruction that enterostomy not only may be life-saving but also may be frequently the only operative procedure necessary.

When a very early diagnosis of obstruction is made and obstruction successfully relieved by operation, much of the treatment outlined above may be unnecessary. In very late cases all treatment may be of little avail. In an active surgical practice there are sure to be all types of obstructive lesions and to say that any one outline of treatment applies to all would be folly.

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FACTORS DETERMINING THE SELECTION OF OPERATION IN OBSTRUCTION OF THE SMALL INTESTINE

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THERE is no more severe test of a surgeon's ability than in the decisions he makes regarding the treatment of intestinal obstruction. The factors which influence his judgment in any given case are many and varied. The general condition of the patient, the time since onset, the cause of the obstruction, the level of the obstruction, the local condition of the obstructed bowel, the changes in the chemistry of the blood—all must be given serious consideration. Then, in addition, there remain certain intangibles which the older surgeon cannot define but which he recalls from past experience of similar circumstances.

The general condition of the patient at the time of entry is, without doubt, the most important single factor in determining what type of operation, if any, can be tolerated. The patient's condition is essentially the resultant of all the factors mentioned in the first paragraph above. Under certain circumstances, the combinations of these factors have led to such a state that a lethal outcome cannot be avoided by any therapy at present available. Hence it becomes a prime necessity for the surgeon to be able to estimate how sick his patient really is.

There will be little difficulty in the very late toxic patient. He will be either in the stage of exhaustion with a grayish pallor; darkly circled, sunken, dull eyes; anxious, drawn, fatigue lines about the mouth; drowsiness; fecal odor to the breath; overflow type of regurgitant vomiting; distended, silent abdomen; and cold, clammy extremities: or in the stage which just precedes it, when his pulse will be rapid, his temperature high, and his face flushed and unhappy. He may be restlessly thrashing about the bed when the painful cramps have not yet given way to the paralytic stage. In neither of these stages is operation likely to be of any value.

In the early stages within a few hours after the onset of symptoms, there will be also little difficulty in making a decision. There has been but slight change in the patient's general condition at this time. The only real danger is from deciding that nothing serious is wrong. The patient has had crampy pains followed by repeated vomiting. Examination gives practically no information, unless visible peristalsis hap-

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pens to be noted or borborygmi are picked up by the stethoscope. The white blood count frequently will be elevated, but the absence of tenderness or spasm tends to give the physician a false sense of security. No trouble will occur for a period of hours depending upon the level of obstruction. Then, there will be a repetition of the attack. This latent period of freedom from symptoms often throws the unsuspecting physician off his guard and retards his diagnosis. The latent period results because of the time that it takes for the bowel above the obstruction to again become filled with secretions. In the objective obstructed or strangulated hernias, of course, there are no difficulties of this kind.

It is in the patients who have passed the first forty-eight hours from the onset of their obstruction that it is exceedingly difficult to make a prognosis. The small bowel usually has distended quite considerably by this time. The contents within it have become foul, stagnant, and toxic. There is no way for the surgeon to decide with certainty whether he is dealing with a strangulated loop or not. The temperature may be normal or subnormal; and the pulse may be slow. It is possible to judge that the patient is beginning to get toxic by manifestations of discomfort and a worried expression. There may be a slight flush at times. The patient becomes restless. He may drowse off for periods only to rouse and thrash about. The tongue looks red and dry. The vomitus begins to get a fecal odor. The white blood count is high—out of proportion to the abdominal signs. The inexperienced surgeon is more likely to underestimate the severity of the condition of his patient than to really evaluate it. He is surprised to find how high the nonprotein nitrogen of the blood is recorded in the chemical laboratory. If he operates, he may undertake a more extensive procedure than is necessary. He should remember that usually following operation the general condition will be worse for some hours, as can be observed by changes in the temperature, pulse, and chemistry of the blood. It is just here that the surgeon of experience can tell that the patient is much sicker than he appears to be. He will not be able to state why he thinks so, if pressed by his colleagues. If the patient has gone beyond the forty-eight- to seventy-two-hour period without manifestations of grave toxicity, it is usually an indication that strangulation is not present, and that the loops are being successfully decompressed either through vomiting or suction drainage. It is well, however, for the surgeon to keep in mind in the early period of an obstruction that he probably is dealing with a strangulation as he has no means of knowing it except by exploration (Case 1).

If the diagnosis of complete intestinal obstruction can be made at the onset of the symptoms, the sooner surgery is undertaken the better.

There should be no mortality from these early operations. It is the ideal time for operation in intestinal obstruction. There are many reasons why operation is safer at this time. The principal reason is that there has been little change in the patient's general condition so that operation is performed under better circumstances. The loops have not become distended to any degree so that they do not get in the way. The obstruction is easier to find and to handle. There is less danger of impairment of the circulation to the bowel. There is less loss of fluids and salts. The bowel can be manipulated more safely without fear of rupture. Spinal anesthesia can be selected unless contraindicated for other reasons with fear of tearing a damaged loop by its use. The recovery after operation in these cases should not be different from that which follows any other abdominal operation.

It may be proper to ask what is the safe period of time to watch a patient without risking a gangrene of the bowel if its circulation happens to be cut off. This is a difficult question to answer. There is really no surely safe period for observation. In an occasional instance, the bowel will be damaged irreparably after three hours of strangulation of its blood supply. On the other hand, a complete cutting off of the circulation may be tolerated for as long as from six to ten hours. There have been cases where the circulation must have been shut off intermittently for much longer periods with eventual recovery of the bowel. Owing to the uncertainty, it is wiser to play on the safe side and not set any limit but to proceed as soon as is expedient. Also, this uncertainty is a very powerful reason for not attempting to reduce an obstructed hernia under average circumstances.

The surgeon is not often called upon to treat intestinal obstruction in the ideal time period, i.e., within the first six to twelve hours. It is usually his lot to have to deal with much later obstructions. If the first forty-eight hours have passed, time should be taken to get the patient into better condition if possible. This can be done by decompression of the obstructed loops by the use of Wangensteen suction drainage. The blood chemistry can be studied and the deficiencies made up as indicated. Salt, sugar, or blood can be given; and drugs so that the patient can be rested until ready for surgery.

When the cause for the obstruction is obvious, the prognosis is usually good. For this reason, obstructed hernias are recognized quickly and operation is done in the favorable period. The same is true for intussusception in children, unless there is doubt about the diagnosis. It is also fairly common to get a reasonably early diagnosis in patients who have obstructions about old adhesions from previous abdominal operations. When the diagnosis is obscure, delay is frequently responsible for a poor prognosis. In consequence, the obstructions due to mesenteric thrombosis, intussusception in adults, volvulus about a

Meckel's diverticulum, obstructions of loops through tears in the mesentery, and internal herniations fall into this latter group. Strangely enough the obstructions which follow recent surgical interventions rarely are picked up early by the surgeons in attendance. The vomiting in the first week or ten days after operation is assumed to be natural for certain patients. The crampy pains are believed to be gas pains only. It is hard for the surgeon to realize that such a calamity can follow one of his operations. The diagnosis thus is made with reluctance. On the other hand, obstructions in the presence of peritonitis are expected. If such a patient passes the two weeks' period without obstruction, it is considered fortunate. Both these latter type obstructions offer a fairly favorable prognosis, however, for frequently it is possible to carry them on suction drainage until the adhesions are absorbed. The high obstructions about the mesentery in infants are usually partial; they constitute a class in themselves, requiring special handling. Obstructions occurring in pneumococcic, streptococcic, or tuberculous peritonitis are conditioned by the severity of the general infection (Case 2).

Generally, it is thought that high obstructions are more dangerous than low ones. From experimental work it has been concluded that the nearer the obstruction to the bile ducts, the more severe is its character. It is our experience that this is not so unless a short high loop is strangulated. The high obstructions are more easily controlled by suction drainage than the low ones. The salt and water balance constitutes the main difficulty in the high obstructions. This can be restored quite readily, after which the toxicity as measured by nonprotein nitrogen approaches normal. The low obstructions sometimes also do well, but there is more opportunity for some loops to twist and become trapped. The plasma volume must be taken into consideration under such circumstances.

The condition of the blood supply of the bowel is of the utmost importance. Strangulation with gangrene modifies the prognosis more than any other single factor as far as surgery is concerned. The surgeon can only hope that the bowel segment will recover when once its circulation has been released from the obstruction. Under hot packs, the bowel should be watched for fifteen to twenty minutes. Signs usually regarded as favorable for viability are: return of reddish color, return of pulsation in the vessels, appearance of a glistening surface on the visceral peritoneum, and peristalsis on stimulation. If the loop does not show these changes, it is usually safer to resect it. Resection should be carried well beyond the damaged bowel into healthy tissue to avoid small thromboses in the vessels. The more extensive the resection is, the more difficult the surgery, with conse-

quently more damage to the patient in shock, etc. Even so, as much as twelve to thirteen feet of bowel, amounting to over half of the small intestine, have been resected with recovery.

When the bowel appears to be able to survive, it should be returned to the abdominal cavity. There is always an element of danger in doing this as is evidenced by Cases 3 and 4 which were handled by experienced surgeons and thought to be viable.

Aside from the blood supply, the amount of distention in the small bowel is of importance. This is more so from the recovery standpoint than from the influence on the type of surgery. It is amazing how much the small bowel can be stretched and yet recover its function. There is, of course, a certain amount of paralysis from overstretching the musculature. This causes a degree of paralytic ileus which must be taken into consideration with every severe, long-standing small bowel obstruction. The damage may be permanent in some cases, but some degree of slow recovery is the rule.

The surgeon who can follow the chemistry of the blood in his patients gets information of great value. He finds that the strangulation obstruction cases do not suffer so much from salt loss as they do from loss of plasma volume. Thus he makes ready for transfusion instead of giving his patient too much salt which may be harmful. The patients who lose many of their upper intestinal secretions through vomiting, on the other hand, require restoration of salts and water. When once the bowel begins to resume its function, blood chemistry studies need not be carried further.

There are certain surgical principles which should be applied to any case of small bowel obstruction. The surgeon should operate only under circumstances as favorable as they can be made. Nothing more should be done than is absolutely essential to restore the bowel to its normal relationships. The removal of an appendix or a Meckel's diverticulum is usually unnecessary and subjects the patient to an added risk when he already has about all that he can care for (Case 1). The simplest procedure which will relieve the situation is the best surgery. Gentleness in manipulation of the bowel will diminish the postoperative discomfort. If the surgeon will trace the collapsed loops upward from the ileocecal region to the distended intestines, he will do less damage to his patient. When a loop with a damaged blood supply is found, after release it should be wrapped in warm gauze packs and observed. If it appears to be recovering, often it may be returned with more safety than it can be resected. In case of doubt, resection should be done. Gangrenous patches can be turned in locally without a more elaborate procedure. Massive gangrene of a loop forces the hand of the surgeon and makes him do more extensive procedures than he would choose under the circumstances. The sur-

geon should have the ability to adapt himself to whatever he finds. Sometimes a sidetracking operation will be the most simple solution of a complicated situation. Care should be taken to minimize trauma and to cover raw areas so that future obstructions may be prevented. At times in chronic recurring obstructions, it will be necessary for the surgeon to use all his ingenuity and versatility in attempting to solve the problem (Case 5). When obstruction has occurred repeatedly about the same area of the intestine, it is sometimes the best surgery to perform a radical resection of this region. The tendency to repeatedly obstruct is usually based upon a pathologic process. Removal of the diseased tissue in such a case is the best insurance against future obstructions (Cases 4 and 5). The type of anesthesia best adapted to the individual should be chosen. It is fortunate that there is now such a wide latitude of choice. When spinal anesthesia can be used, it makes the surgery much easier from a technical standpoint. The inhalation anesthesias are to be avoided whenever it is possible without sacrifice.

Six case reports are appended to illustrate problems which may arise in the selection of the proper care for patients with obstruction of the small intestine.

CASE 1.—C. E., No. 123661, a boy seventeen years of age, was admitted to the Rochester Municipal Hospital on January 20, 1937. Six days previously he had a sore throat not accompanied by chills or fever. The following day there was a constant severe abdominal pain around and above the umbilicus. The bowels failed to move and numerous enemas were not effectual. He had vomited three to four times daily since the onset of pain. The pain was not crampy in character. One year previously he had had a gastrointestinal upset but except for frequent constipation, no other trouble referable to the gastrointestinal system. His father had died of pulmonary tuberculosis three years before, but he had not been exposed to it. He worked hard himself and did not get enough sleep. He fatigued easily. He had felt run down for about one year. For one month he had had night sweats. There had been no cough or afternoon fever.

Examination showed temperature, 37° C.; pulse, 70. He did not appear ill, although he was dehydrated. The throat showed acute tonsillitis and pharyngitis. The abdomen was markedly distended, tympanitic, and tender in all quadrants on pressure. Intestinal patterns were visible, and borborygmi were heard by some observers but not by others. A fluid wave and shifting dullness were noted. The white blood count was 15,050. Some observers thought that he looked acutely ill. There were comments in the notes on his restlessness. Other observers thought that he was drowsy. The general impression was that of discomfort and worry on the part of the patient.

He was placed on duodenal tube drainage. The fluid drainage from the tube had a decidedly fecal odor. Temperature was recorded as subnormal, and pulse remained at 70. The nonprotein nitrogen of the blood was 66, and the chlorides were 386 mg. per cent. Flat plate of the abdomen showed enormously distended loops of small bowel filled with fluid, definite levels being depicted. He was given intravenous salt and glucose. At operation under spinal anesthesia, volvulus was found to have occurred about the fibrous cord of a Meckel's diverticulum completely obstructing the small bowel down to that level. Upon division of the cord, deep vigorous peristalsis occurred in the obstructed bowel. The diverticulum was removed and the base in-

verted. The obstruction was relieved by the passage of the intestinal contents while on the operating table. Transfusion was done following operation; also, fluids were supplied. Suction was continued. The nonprotein nitrogen had dropped to 50 on the next day, and the chlorides were 574 mg. per cent. On the second day postoperatively, the nonprotein nitrogen was 37 and the chlorides 491 mg. per cent. Suction drainage was discontinued. Temperature which had risen to 38.5° C. dropped slowly to normal during the week. His bowels moved spontaneously or by enemas daily following operation. He had a smooth convalescence and was discharged well on February 4, 1937.

This case illustrates the difficulty in arriving at a diagnosis when there are other possibilities to consider. He was seen by several surgeons before the diagnosis was made. Even then his condition was thought to be better than the blood chemistry studies showed it to be. At operation, the surgeon relieved his obstruction by dividing the fibrous cord of the Meckel's diverticulum. He could not resist the temptation to remove the diverticulum. This is meddlesome surgery in the presence of acute obstruction. It exposed the patient to the danger of a peritonitis. The obstruction was relieved when the cord had been divided. The removal of the diverticulum added a risk which should not have been taken.

CASE 2.—S. V., No. 83048, a girl, ten years of age, was admitted to the Strong Memorial Hospital on November 27, 1933. She had been well until November 24, 1933. At this time, she developed a severe chill and headache. This was followed by spasmodic abdominal pain. The pain was low in the midabdomen and excruciating in character. There had been no nausea or vomiting. Upon examination, it was apparent that she was acutely ill and toxic. Temperature reading was 104° (40° C.) and pulse rate, 146. The tonsils were reddened. The abdomen was very tender to palpation in both lower quadrants. There was slight spasm of the abdominal muscles but no real rigidity. There were undoubted signs of peritoneal irritation but no evidence of perforation of a hollow viscus. The urine was loaded with pus. There was a slight vaginal discharge. The white blood count was 35,000. The degree of prostration and the general reaction seemed out of proportion to the peritoneal condition. This indicated that the abdominal signs were merely part of a generalized process, either a streptococcic or a pneumococcic peritonitis. It was decided to do an abdominal paracentesis to see if the type organism could be recovered. Otherwise she was to be watched and to be given supportive treatment. Laboratory examinations showed throat culture, 50 per cent streptococci; vaginal culture, *Streptococus hemolyticus;* repeated blood cultures, no growth; peritoneal fluid, *Streptococcus hemolyticus.* She also showed some patches of pneumonia in both lower lung fields within a few days after entry. During the next two weeks she was very ill. She became distended, had nausea and vomiting; also, fluid appeared in both pleural spaces. The pleural fluid was spontaneously reabsorbed, but signs in the abdomen suggested localization of an abscess with beginning intestinal obstruction. Accordingly on December 7, 1933, under avertin and local anesthesia, a McBurney incision was made. The ileocecal valve was identified and a collapsed loop of bowel traced back to some adhesions on the lateral wall of the right pelvis. When these adhesions had been released, the collapsed loops filled with contents from above. There was a marked plastic exudate and about 50 to 75 c.c. of fluid were present. Closure was made without drainage. Postoperatively she made a good recovery except for the localization of abscesses in each thigh where hypodermoclyses

had been given. These abscesses were drained. She went home in good condition on January 12, 1934. She had had no further trouble with her intestinal tract when examined one year later.

This case illustrates the value of being able to estimate the general condition. If the surgeon had operated upon this girl when she was suffering from a general peritonitis, he would have had a fatality without any doubt. Operation would have accomplished nothing. The bowel was in a state of paralytic ileus. Enterostomy, the simplest procedure, would have been useless. By waiting until she had reacted to her infection and had partly overcome it, the surgeon was able to call to his aid all the factors of acquired resistance to the infection. When definite localizing signs of an organic obstruction were present, there was a definite indication for action. The bowel had recovered its tone. Release of the adhesions restored the intestine to its normal relationships and function.

CASE 3.—B. C., No. 119,592, a woman, forty-two years of age, was admitted to the Strong Memorial Hospital on September 22, 1936. One month previously, she had been operated upon for acute intestinal obstruction. At this time she was very toxic after forty-eight hours of crampy pains, obstipation, and vomiting. She was stuporous; and she had abdominal distention, fecal vomiting, and a pulse rate of 130. At operation a loop of small bowel had strangulated about the point of attachment of a previous uterine suspension. The bowel was released. It was quite dark, but the circulation returned so that it appeared adequate. She recovered under suction drainage until the third day. Pneumonia developed then, and it was necessary to keep her in an oxygen tent for twelve days. On the fifth postoperative day, her wound began to discharge. The local physician opened it wider. It has discharged fecal material since then. Two weeks before entry she began to run fever. A fullness developed in the right lower quadrant. Upon admission, an abscess in the right lower quadrant was drained through a McBurney incision. There was a finger-sized opening in the bowel at the bottom of the abscess cavity. Following drainage the abscess cavity obliterated, and the fecal fistula gradually became smaller. It finally closed.

This patient illustrates the danger inherent in returning a strangulated loop, which appears to have recovered. The surgeon used good judgment at the operation as the patient was in a critical condition. But some portion of the bowel failed to live. The resulting fecal fistula and localized abscess may be responsible for future trouble.

CASE 4.—E. W., No. 109272, a woman, forty-three years of age, was admitted to the Strong Memorial Hospital on December 4, 1935. There had been a sudden onset of severe crampy pains in both lower quadrants twenty-four hours prior to admission. There was temporary relief following the passage of four to five loose stools but a return of painful cramps about ten hours later. The cramps became worse as time elapsed. There was nausea but no vomiting. She had passed a little gas with an enema at the onset of the attack but none after that. In her previous history, there had been three laparotomies for pelvic and appendiceal disorders. Seventeen years ago on the eighth day after a cesarean section, her bowel had obstructed, necessitating an operation for release of adhesions. Since that time she had been free from

symptoms. Upon examination at entry, she appeared to be in moderate pain. There was distention of the abdomen. Peristalsis with a definite small bowel pattern was visible in the right lower quadrant. Many abnormal peristaltic sounds were easily heard through the stethoscope. The temperature was 37°; pulse, 76. The white blood count was 12,000. A scout film of the abdomen showed a loop of small bowel distended with gas in the right pelvis. A duodenal tube was inserted and 400 c.c. of greenish fluid with a moderate amount of gas was recovered. There was some relief from this procedure, but the white blood count rose to 16,000.

At operation two hours later, there was a large amount of slightly cloudy nonodorous fluid in the peritoneal cavity. Several loops of small bowel were bound down in the pelvis by long dense fibrous adhesions, stringy in character. These were released. When this had been done, it was apparent that there was a badly obstructed loop of a deep purplish plum color buried under a mass of adhesions in the pelvis. These adhesions were attached to the posterior wall of the bladder and to some remnant of her pelvic organs. When the adhesions had been dissected from the bladder, it was possible to bring up the intestinal loop. The obstructed bowel was about 12 inches (30 cm.) from the ileocecal valve. Although the discoloration of this loop was considerable, in about ten minutes under hot gauze packs it became reddish purple, and pulsations could be seen returning in the vessels. There were no areas of frank necrosis. The bowel wall glistened and peristalsis could be started by gentle pressure on the segment. Accordingly, it was decided that the loop was viable. Closure was made. Recovery from operation was quite satisfactory. The day after operation the nonprotein nitrogen had fallen from 38 to 28 mg. per cent. She was discharged to her local doctor on the tenth day after operation. The wound was well healed. A smooth nonresidue diet and the use of mineral oil were recommended. She was readmitted twelve days later because of recurring abdominal pain. Two days previously there had been a sudden onset of sharp crampy pain in the right lower quadrant. This passed off but returned again twenty-four hours later. Four hours before admission it became quite severe. She again presented the signs of an acute intestinal obstruction. At operation, the ileum was found densely adherent to the anterior abdominal wall about 10 to 12 inches from the ileocecal valve. These adhesions were dissected free; in doing so, a small quantity of clear, odorless fluid seemed to come from a perforation in the loop. Accordingly, the involved loop which was narrowed and scarred by fibrous adhesions was resected and an end-to-end anastomosis made. Examination of the resected segment showed a dusky narrowed circular fibrosed area with a small opening into the lumen at one side. The mesentery below was black. There was edema of the wall and thrombosis of both large and small vessels. Apparently the segment which had appeared viable at the previous operation had become necrotic at one point but fortunately had been walled off by adhesions.

Recovery from this operation was uneventful. Since discharge from the hospital there has been no further trouble.

Note.—This sequence of obstructions represents the danger in returning a badly damaged bowel to the abdominal cavity. According to our present criteria, there was every reason to suppose that this segment would recover. Apparently, however, one portion of the gut had progressive thrombosis of its vessels with necrosis, perforation and walling off by adhesions of the involved area. The question may be raised as to whether a badly damaged segment is not always a potential danger either for perforation or as a focus for future obstruc-

tions. Unless the inflammation completely resolves, it leaves an area of damaged intestine with chronic inflammation, edema and fibrosis, and the formation of protective inflammatory reactions in the neighborhood. This furnishes an ideal background for another obstruction.

CASE 5.—M. D., No. 38839, a woman, nineteen years of age, was admitted to the Strong Memorial Hospital on October 20, 1931. She was operated upon for supposed acute appendicitis, but instead calcified lymph nodes were found. One of these was removed. Microscopically, it showed old tuberculosis. About one month later she began to have attacks of crampy pains, vomiting, distention, and joint trouble. The attacks were intermittent in character. There would be days of complete relief. She was followed closely in the hospital for about six weeks, two conditions being seriously considered—tuberculous peritonitis and partial intestinal obstruction. The attacks became progressively more severe until it became necessary to explore. Dense adhesions were found almost completely obstructing the midileum. There were also other loops kinked and twisted by adhesive bands between them. At one point a small volvulus had occurred about an adhesion. All these conditions were corrected. The abdominal scar in this patient was keloidal in nature and the tendency to form scar was marked. One week later because of failure to improve, she was operated upon a second time. The loops of bowel were densely matted together at the lowest point of obstruction previously noted. Even when these loops had been released there was so much scarring in the mesentery that they tended to resume their old kinked relations. Accordingly a lateral anastomosis was made around them. After a slow recovery and a prolonged convalescence during which she had complaints referred to the gastrointestinal, neurologic, and genitourinary systems, she was discharged from the hospital on February 28, 1932. She continued to have pains in the joints but had no further gastrointestinal upsets until October 13, 1932. At this time she had an attack of abdominal pain and vomiting which progressed to complete obstruction. Exploration on October 15, 1932, showed many adhesions and a band obstruction rather high in the jejunum well above the anastomosis. At closure, 50 c.c. of amfetin was left in the peritoneal cavity in the hope of preventing adhesions. She gradually recovered and was discharged on November 1, 1932, free from complaints. She was readmitted two days later with a return of symptoms. Upon failure to improve, the abdomen was explored again on November 5, 1932. Many adhesions were present involving 3 or 4 feet of the terminal ileum in kinks, much of the trouble being due to adhesions across the mesentery.

For the next two months she continued to have discomfort alternating with periods of freedom. The condition, however, gradually went on to complete obstruction. On November 20, 1932, for the fifth time operation was done because of obstruction. A mass of pathologic small bowel loops 3 feet in length was resected and an end-to-end anastomosis made between healthy jejunum and terminal ileum. A broad piece of rubber dam was left between the undersurface of the incision and the bowel. It was brought out as a drain at the lower end of the wound. It was hoped in this way to prevent adhesions to the peritoneal side of the incision. The excised specimen showed a greatly thickened mesentery with subacute inflammation of the bowel. There were no tubercles or epithelioid cells identified. She began to have obstructive signs once more about three weeks after operation. The sixth operation, on December 14, 1932, showed a definite obstruction at the site of the end-to-end anastomosis. The bowel was tied in a complicated inflammatory reaction at this point. The simplest way to relieve the obstruction seemed to be by a lateral anastomosis between the dilated ileum above the obstruction and the ascending colon. This was done. She improved until January 20, 1933. At this time she had diarrhea, vomiting, abdominal cramps, and signs of an imminent obstruction. Conservative measures were adopted—

changes in diet, atropine, ultraviolet light, paregoric, opium, bismuth. For one month she required very careful nursing, but at the end of that time her intestinal tract began to behave normally. She was discharged to a convalescent hospital where she slowly recovered. After six months in this institution, she returned to her home. In December, 1933, she had gained 24 pounds and felt normal in every way. Her bowels were regular. There was no further pain in the abdomen. She has remained in excellent health since that time without any further complications. She has taken her place in the community for three years and has not lost any time because of illness.

This case shows how many different lines of attack a surgeon must call upon in order to get his results in some complicated cases. It also illustrates the value of courage and optimism on the part of both patient and attendants.

CASE 6.—J. B., No. 18198, a man, forty-two years of age, was admitted to the Rochester Municipal Hospital on September 20, 1928. He was a very sick man with a generalized peritonitis from a ruptured appendix of several days' duration. The appendix was removed, no drainage was instituted. Paralytic ileus developed, and a jejunostomy was performed under local anesthesia. After one week of continued paralytic ileus, the jejunostomy began to function. One week later, a pelvic abscess was drained in the right side. Three weeks later because of failure to improve, exploration of the pelvis was done. Many adhesions were present, and some definite points of obstruction were freed. This resulted in normal functioning of the bowel. Then the enterostomy began to be troublesome; much fluid was lost through this opening. The abdominal wall became excoriated and required constant attention. On November 12, 1928, the enterostomy opening was resected and an end-to-end anastomosis was done. His recovery was rapid following this. He was discharged on December 10, 1928. He was readmitted for an acute intestinal obstruction with volvulus on December 19, 1928. This had followed eating a large meal at home. After release of this obstruction, he made a good recovery and was discharged on January 12, 1929. He had no further trouble from obstruction. A ventral hernia was repaired in June, 1931. Since that time he has been in the dispensary for many minor complaints. He has had no further intestinal involvement to date.

Enterostomy for paralytic ileus is not a good procedure. The bowel will not drain well until it recovers its tone. In the presence of an acute peritonitis, enterostomy is often a positive danger. Wangensteen suction drainage will usually do everything that an enterostomy can. The patient is not subjected to a major risk by this latter type of therapy.

HIRSCHSPRUNG'S DISEASE; INDICATIONS FOR AND RESULTS OBTAINED BY SYMPATHECTOMY

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THE term Hirschsprung's disease[2] is synonymous with congenital megacolon of neurogenic origin and is used to distinguish this disease from acquired megacolon arising from chronic obstructing lesions. In the former, the megacolon results from neuromuscular dysfunction; whereas, in the latter, it develops from mechanical obstruction. The correction of Hirschsprung's disease therefore becomes a neurosurgical problem, a problem of reestablishing normal peristaltic movements and regulated periods of defecation.

ETIOLOGY

Hirschsprung's disease is presumed to be of neurogenic origin, in which the mechanism for filling the intestine or for retaining intestinal content overbalances the emptying mechanism; this accounts for the retention of feces in the dilated and hypertrophied sigmoid and colon (Fig. 1). The patients are usually children or young people. It is apparent that the inhibitory muscular responses to the colon, and the contracting stimuli to the internal sphincter of the anus from the sympathetic outflow are more powerful than the motor responses to the musculature of the colon and the inhibitory responses to the internal sphincter of the anus, which come from the parasympathetic nerve supply. It is difficult to explain this dysfunction of the neuromuscular mechanisms, unless the subject is constitutionally inferior, and the general nervous system calls for hyperactivity of the sympathetic nervous system to meet the demands of the body in endocrine secretions, which in turn develop excessive stimuli in the sympathetic fibers innervating the colon and internal sphincter of the rectum. Defective parasympathetic innervation might produce a similar effect, since the emptying stimuli would be less than the filling and retaining stimuli.

Acquired megacolon results from chronic obstruction which is seen to result from elongation of the mesentery, torsion of the segment or multiplication of the intestinal loops. Anatomic factors, such as faulty valves, aplasia of the musculature above the rectum, and obstructing bands, are likewise responsible for megacolon. Infective processes in-

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volving the intestinal musculature and slowly growing carcinomas of the rectosigmoid may also give rise to chronic obstruction and produce megacolon.

PATHOLOGY

The pathologic changes of megacolon of neurogenic origin, and of obstructive origin, are very similar. Hirschsprung, in 1886, in describ-

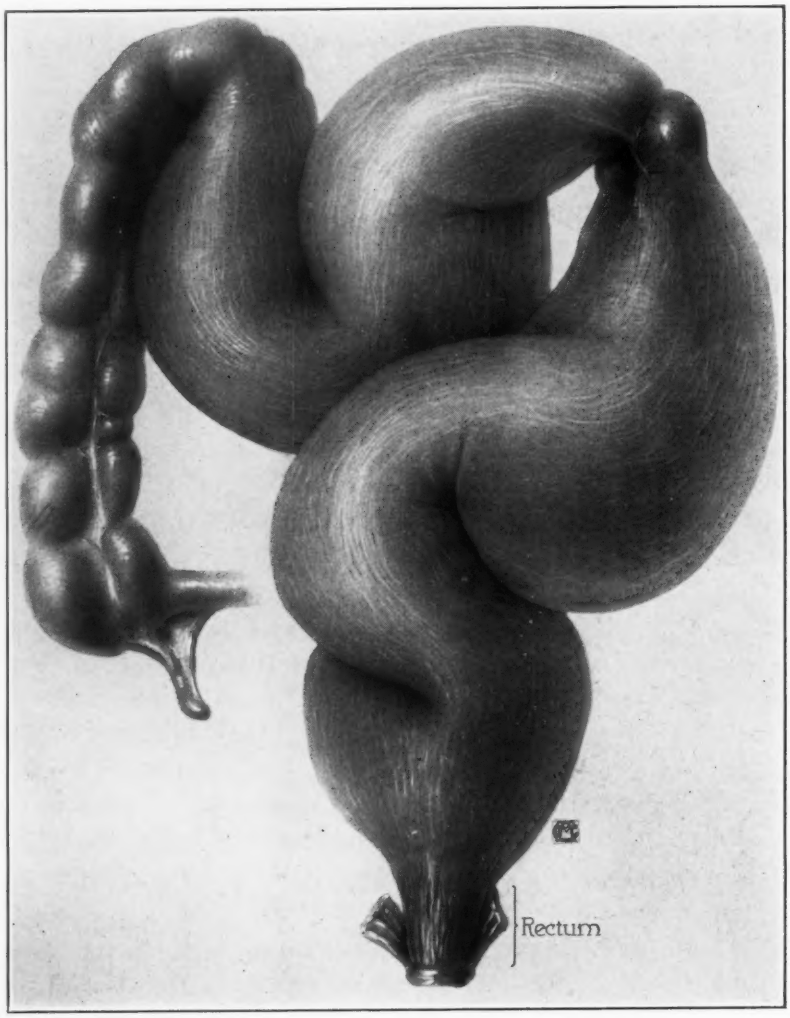

Fig. 1.—Drawing made at the time of operation showing megacolon due to Hirschsprung's disease. Note the portion of bowel involved and the absence of mechanical obstruction.

ing the condition found in children, referred to the disease as a congenital, high-grade dilatation of the colon with thickening of all tunics, especially the tunica muscularis, with retention of large quantities of fecal matter. The congenital type differs from the acquired type in that the former type is present at birth, whereas the latter develops subsequently. In the congenital type of megacolon the left half of the colon

and occasionally the entire colon is enlarged without the presence of any obstructing lesion, while the acquired type of megacolon is always associated with a demonstrable mechanical obstruction. The acquired type of megacolon results from chronic obstruction and not from acute obstruction, which produces dilatation of the colon with thinning of the tunics.

Thickening of the tunics, particularly the muscular layer, appears to be a compensatory response to overcome the obstruction, whether it is neurogenic or mechanical (Fig. 2). Dilatation is but another com-

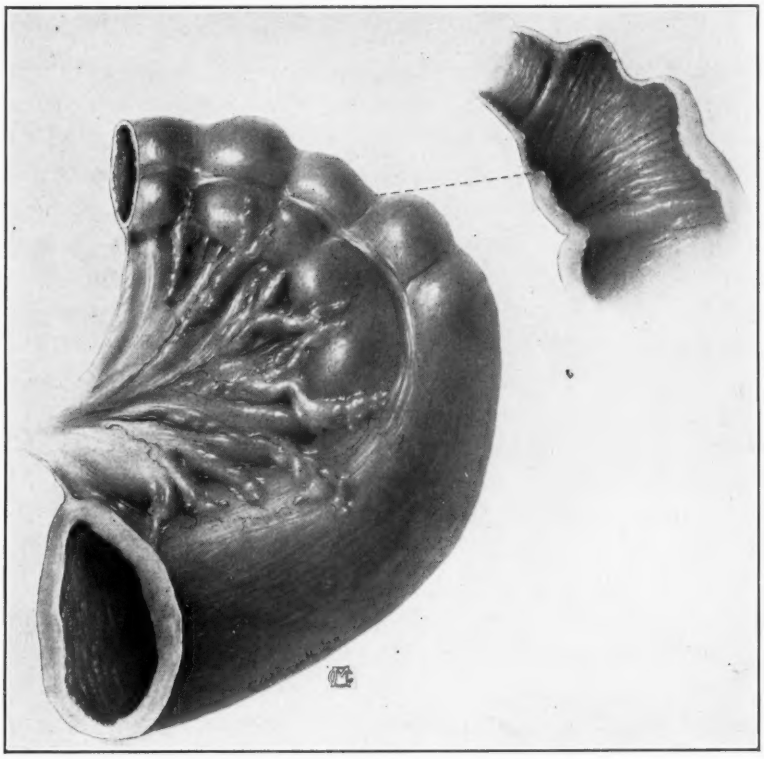

Fig. 2.—Showing the absence of mechanical factors limiting the proximal dilatations of the transverse colon.

pensatory factor in providing for the retained feces and accumulation of gas. Histologically, the circular and longitudinal muscular fibers are seen to be hypertrophied, which explains the elongation of the sigmoid mesentery. The peritoneum covering the enlarged bowel has lost its luster and often reveals scarred striae running parallel with the colon, causing it to appear as if it were in a fibrous bag. Lymphatic channels may be seen to run underneath this peritoneal covering and are often associated with increase in number and enlargement of lymph nodes. The submucosa and the mucosal layers are likewise hypertrophied and may be associated with ulcerated areas.

SYMPTOMS

The symptoms arising from Hirschsprung's disease may vary from obstipation to complete obstruction. In the milder cases the results may be only abdominal distention and infrequent defecation; whereas, in the more exaggerated forms, marked abdominal distention with flaring of the ribs, respiratory and cardiac embarrassment, and toxic symptoms from retained feces, with absence of spontaneous defecation, may occur. The toxic symptoms result in general malaise, lack of appetite, emaciation, and irritability with a septic type of temperature.

MEDICAL CARE

In the milder cases, the condition often can be controlled by a medical regimen which consists of administration of mild cathartics, such as milk of magnesia, mineral oil by mouth and by retention enema, and the occasional use of glycerin suppositories. In the more severe cases, the diet may need to be changed from bulky foods to a diet containing nonresidue foods, with the substitution of broth and of preparations of milk and sugar to maintain the calorie value. Medication with such substances as acetylcholine, pituitrin, eserine (physostigmine), and drastic purgatives are often used. The distended colon may require emptying by mechanical means after thorough dilatation of the external sphincter. If oil and soapsuds enemas are ineffective, enemas containing hydrogen peroxide may become necessary to break down fecal impactions. Hospitalization is necessary to establish a routine, and it is only when these regulated measures, carried out at home, fail, that surgical intervention is considered.

Diathermy when applied to the abdomen has produced an increase in peristaltic movement in evacuation of fecal content, but unfortunately, the effects are temporary and the results do not continue when diathermy is discontinued.

Surgical procedures such as appendicostomy, colostomy, enterostomy, and partial colectomy were formerly employed in severe cases; but since so many of these procedures failed to give relief and were attended with a surgical mortality of 25 per cent or more, the newer method of operation on the sympathetic nerves of the pelvis has replaced the major operations.

ANATOMY AND PHYSIOLOGY OF THE RECTUM AND BLADDER

Gaskell[12] has pointed out that there are three sphincter muscles which terminate the different regions of the primitive intestine: (1) the ileocolic sphincter at the end of the small intestine; (2) the internal anal sphincter at the end of the coprodeum; and (3) the internal vesical sphincter and the urethral muscles at the end of the urodeum. The motor nerve cells for the ileocolic sphincter are situated in the superior

mesenteric ganglion; whereas, the motor nerve cells for the internal anal sphincter and the internal vesical sphincter are in the inferior mesenteric ganglion.[10, 11, 15] The inhibitory nerves to the internal anal sphincter and to the internal vesical sphincter, according to Elliott,[10, 11] travel through the pelvic nerve as a part of the sacral outflow. The motor nerves to the musculature of the large intestine, according to Bayliss and Starling,[5, 6] and also according to Langley and Anderson, are likewise a part of the sacral outflow and travel through the pelvic nerve. Elliott[9] has confirmed this by determining that injection of epinephrine does not cause contraction of any part of the large intestine, with the exception of the internal anal sphincter. Inhibition of the musculature of the large intestine is mediated through inhibitory nerves, the cells of which are in the inferior mesenteric ganglion;[10] stimulation of the lumbar splanchnic nerves, or of the hypogastric nerves, causes relaxation of the musculature of the colon.

Learmonth and Markowitz[18, 19] have shown that electric stimulation of the presacral nerve and of the inferior mesenteric nerve of the dog leads to dilatation of the colon and to contraction of the internal sphincter of the anus. They also demonstrated that section of these nerves increased the activity of the lower portion of the colon.

To clarify further the anatomy involved, it probably would be well to review the anatomic arrangement of the sympathetic fibers which produce inhibition of the musculature of the lower portion of the colon and constriction of the internal sphincter of the anus. The postganglionic sympathetic fibers which carry these impulses originate in the intermesenteric plexuses. This network of nerves descends on the anterolateral aspect of the abdominal aorta, from the level of origin of the superior mesenteric artery downward. On each side there are two or three large trunks, which arise from: (1) the semilunar ganglion and the celiac plexus; (2) an anastomotic loop which crosses the aorta transversely, below the origin of the superior mesenteric artery; and (3) the aorticorenal ganglion, or the renal periarterial plexuses.

The intermesenteric plexus is joined on each side by branches from the first and second lumbar ganglia. The branches contain myelinated fibers; those on the right side pass between the vena cava and the aorta to reach the anterior side of the aorta. The fibers which form the intermesenteric plexuses are thus derived from two sources: their original fibers spring from that part of the abdominal sympathetic system which is connected with the thoracic splanchnic nerves; whereas, the branches which the plexus receives as it descends along the aorta spring from the lumbar ganglia or trunks.

According to Elaut,[8] the triangular mass, the superior hypogastric plexus, receives a series of secondary connections from other regions:

first, from the inferior mesenteric plexus which lies within the pelvic mesocolon at its left; and second, from the last ganglion of the lumbar chain on each side.

Anatomists have held different opinions concerning the extent to which the lumbar fibers mingle with those of the intermesenteric plexus proper. According to Delmas, Laux, and others, the mesially directed lumbar communicating branches, constituting the pelvic splanchnic nerves, remain distinct in the outer part of the plexus and ultimately form the lateral roots of the presacral nerve of Latarjet (superior hypogastric plexus of Hovelacque). On the other hand, Hovelacque held that these lumbar communicating branches actually contributed to the intermesenteric plexus. The point is one of great significance. If the former view were correct, lumbar ramisectomy and ganglionectomy would affect only that portion of the bowel that is innervated through the presacral nerve, the lower part of the rectum, and the internal sphincter of the anus; if the latter arrangement were true, it would affect, but only partly, the descending and sigmoid portions of the colon, as well as the rectum and the internal sphincter of the anus.

Immediately below the level of origin of the inferior mesenteric artery, a large branch leaves the intermesenteric plexus of each side, and passes inward, on the aorta, to reach the inferior mesenteric artery about 1.5 cm. from its origin. Finally, these two trunks unite and give rise to three or four branches which course along the lateral borders of the vessel, communicating at intervals with each other. From these nerves subsidiary trunks arise at levels of the main divisions of the artery. Soon, however, they abandon the vessels and anastomose with one another in avascular parts of the mesosigmoid. From this network the final nerves of distribution are derived; these slender filaments cross the juxtacolic vascular arcades and enter the wall of the bowel between the terminal branches of the vessels, and account for part of the innervation of the colon and internal sphincter; the remaining sympathetic innervation is composed of fibers which continue downward into the pelvis from the intermesenteric plexus and from the lumbar trunks below the second lumbar ganglion, to make up the superior hypogastric plexus before entering the superior hypogastric ganglion where the sympathetic nerves mingle with parasympathetic fibers, and finally is distributed to the musculature of the lower part of the colon, sigmoid, and internal sphincter of the rectum.

SURGICAL CONSIDERATIONS

In comparing the results in treating the various degrees of megacolon by the various types of sympathectomy, it becomes apparent that the more advanced the disease, the more complete the resection of sympathetic fibers must be in order to produce the results desired. The operation of Wade,[22, 23] removal of the first and second lumbar ganglia on the

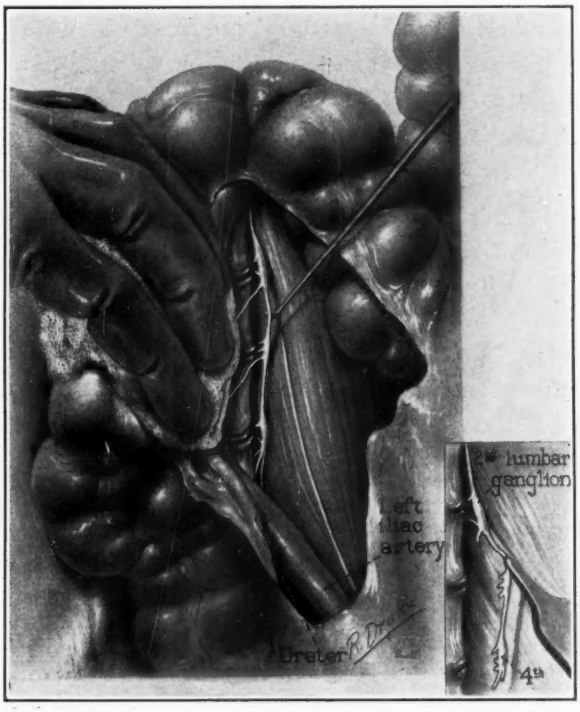

Fig. 3.—Exposure and resection of the left lumbar sympathetic trunk with the second, third, and fourth lumbar ganglia.

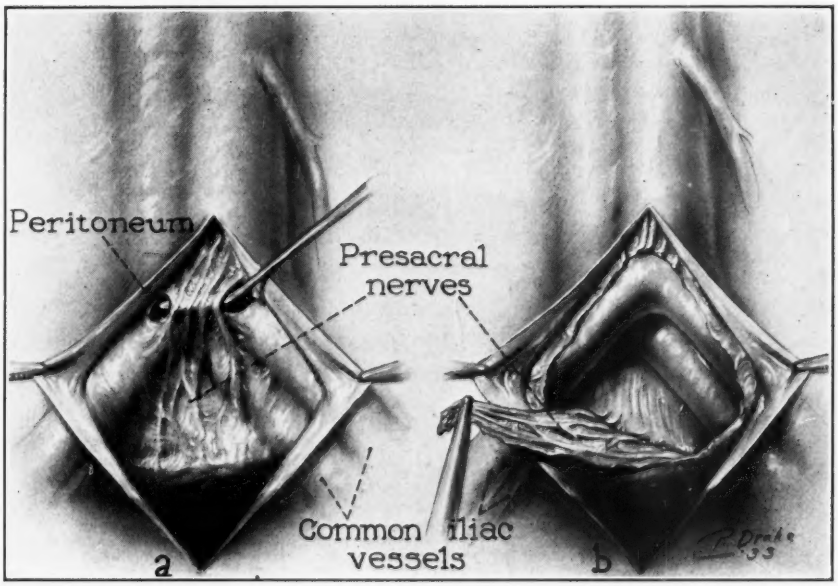

Fig. 4.—*a*, Elevation of presacral nerves as they cross the bifurcation of abdominal aorta; *b*, illustrating the method of presacral nerve resection.

left, is sufficient in the moderately severe case. Bilateral sympathetic ganglionectomy and trunk resection performed by Judd and the author,[14] which includes removal of the second, third, and fourth lumbar ganglia with the intervening trunk, has been found to be still more effective[1] in my experience than unilateral resection (Fig. 3). Resection of the inferior mesenteric nerves in conjunction with resection of the presacral nerve (Fig. 4), the operation of Rankin and Learmonth,[20, 21] has been shown to be effective in the moderately advanced case, but has failed to give all that has been desired, as has bilateral lumbar trunk resection in the most advanced cases of Hirschsprung's disease.

This observation prompted me to increase the scope of the operation still further and it now includes resection of both lumbar trunks, including the second, third, and fourth lumbar ganglia, with wide resection of the superior hypogastric plexus, presacral nerve, situated on the promontory of the sacrum in the triangular space below the bifurcation of the abdominal aorta. This procedure is employed to include all of the sympathetic fibers from the lumbar chain, all of the intermesenteric fibers descending into the pelvis below the inferior mesenteric artery, and those fibers which rejoin the hypogastric plexus from the inferior mesenteric nerve in the mesocolon of the pelvis, thus leaving only a small group of fasciculi which have followed the inferior mesenteric artery and its branches to the lower part of the colon and internal sphincter of the anus.

Although this extensive lumbar, presacral, and inferior mesenteric sympathectomy is effective in relieving the symptoms of Hirschsprung's disease involving the sigmoid and descending colon, it has not been effective in relieving the symptoms when the ascending and right half of transverse colon have been involved, which suggests that inhibitory responses reach the ascending and transverse colon through nerves which accompany the superior mesenteric artery.

This opinion was further substantiated by experiences obtained in resecting the splanchnic nerves and removing the first and second sympathetic lumbar ganglia and intervening trunk on both sides in the treatment of essential hypertension, since these patients occasionally complained of increased intestinal peristalsis following the completing of the operation on both sides. The increased peristalsis resulted in increased daily bowel movements. This frequency soon adjusted itself to one or two daily bowel movements, unless the patient ate generous helpings of fruit such as prunes, figs, or raisins when diarrhea might follow. This increased intestinal activity encouraged me to employ bilateral resection of the splanchnic nerves in conjunction with bilateral removal of the first and second lumbar ganglia in a case of Hirschsprung's disease in which the entire colon of a youth aged nineteen years was involved (Fig. 5). The results were extremely gratifying in that

the patient obtained one to two spontaneous daily bowel movements following the operation. It also encouraged us to employ the same procedure for two female patients who complained of obstinate constipation, attributable to an enlarged atonic colon, with equally good results.

Therefore, it would appear that extensive sympathectomy which includes the splanchnic nerves and first and second sympathetic ganglia on both sides is the operation of choice when megacolon is of neurogenic origin and involves the whole colon. The more limited operations may be employed when the disease is mild in degree and involves only the sigmoid and lower portion of the descending colon.

Surgical Indications and Preoperative Preparation.—As previously stated, surgical intervention is not instituted in the mild cases of Hirschsprung's disease in which medical treatment is adequate, but if it be-

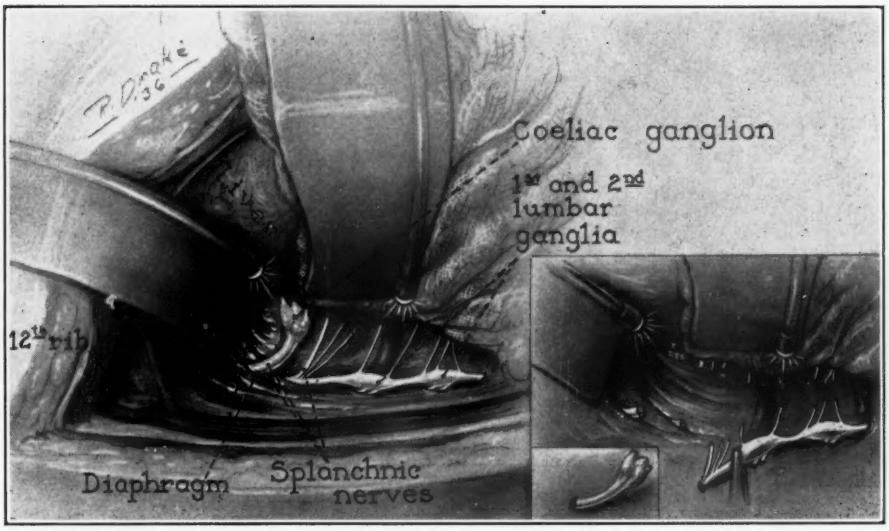

Fig. 5.—Exposure and resection of splanchnic nerves with a portion of the celiac ganglion and removal of the first and second lumbar ganglia.

comes necessary for the patient to return to the hospital more than two or three times for emptying of the colon and the employment of a still more rigid regimen, sympathectomy is indicated. I have operated on a child aged five and a half months, but have observed that the operation is more easily accomplished if the child is one or more years of age. The administration of acetylcholine, which inhibits the activity of the sympathetic nervous system, has served as a preoperative test in selecting patients for sympathectomy.

Before operation is indicated, hospitalization is required and thorough evacuation of the filled colon is necessary, but although this process may require a week or two, the general nutrition of the patient should not be neglected. The colon may be emptied by the procedure employed in following out medical care, and nutrition should be maintained by

means of a high calorie, nonresidue diet with the addition of vitaminized oils. Complete physical examination and laboratory tests are essential to determine if operation can be performed. It is not always wise to employ barium enemas to determine the size of the colon, for often a week or more is required to remove thoroughly all of the barium, which may delay the preoperative period. Roentgenograms made after inflation of the colon usually give the desired information. It is not wise to prolong preoperative preparation unduly for fear of impairing the general nutrition by eagerness to keep the colon empty on a nonresidue diet, since some patients object to taking sufficient food in the prescribed diet to maintain the proper intake of calories.

Technic for Removal of Lumbar Sympathetic Ganglia.—The patient is placed on the operating table in the laparotomy position. The most suitable anesthetic is ethylene and ether or spinal anesthesia supplemented with ethylene.

The incision[4] is made from the symphysis pubis to a point 5 to 7 cm. above the umbilicus, between the rectus abdominis muscles and to one side of the umbilicus. The sheath of the rectus muscle subsequently is opened on each side below the umbilicus and on the left side above the umbilicus, facilitating closure along anatomic lines. If the abdomen is extremely flaccid, it may be advisable to make an overlapping closure (C. H. Mayo type) in the external leaves of the fascia of the rectus abdominis muscle. Before the peritoneum is opened, the patient is lowered from the horizontal position to the Trendelenburg position, thus insuring better exposure of the lumbar sympathetic ganglia. Although general exploration may reveal other abdominal lesions, they are not disturbed at this time, since it is desired to avoid the additional risk of contamination. The intestines are packed upward, as they are when hysterectomy is performed. It is immaterial whether the ganglia are approached first on the right or on the left side. Usually, the ganglia of the right side are more difficult to approach because the intervertebral veins run across the sympathetic trunk. To elevate the inferior vena cava is more difficult than to elevate the abdominal aorta and the common iliac artery on the left.

When exposing the left lumbar sympathetic chain, it is necessary to loosen and elevate the sigmoid and the lower portion of the descending colon. This is done by incising the peritoneum superior and just lateral to the anterolateral border of the upper portion of the sigmoid and the attachment of the lower portion of the descending colon. When the line of cleavage is once started, the large bowel can be elevated readily and can be retracted, with the posterior wall of the peritoneum, beyond the median line. This exposes the ureter (as it courses over the bifurcation of the common iliac artery), the left common iliac artery and vein, the lower end of the abdominal aorta, the genitocrural nerve (which per-

forates the psoas muscle), the psoas muscle, the lumbar vertebrae, the lymph nodes, and the lumbar sympathetic ganglia, trunk, and rami communicantes which lie on the lumbar vertebrae, just mesial to the psoas muscle. The ureter on the left side is more easily retracted mesially than laterally. With a moist sponge it is held gently, together with the colonic mesentery,[1] the upper end of the sigmoid, and the lower end of the descending colon, in position in the median line. The abdominal aorta is elevated and is retracted mesially by traction with a finger on the gauze sponge. This is held by an assistant. The sympathetic ganglia, trunks, and rami are then dissected free by a wet cotton ball dissector held in thumb forceps. It is well to begin at one or the other end of the lumbar sympathetic chain. On the left side, it is preferable to expose the fourth lumbar ganglion at the brim of the pelvis and to divide the sympathetic trunk below it. All of the rami, including those to the spinal nerves, the hypogastric plexuses, and the aortic plexuses, are then divided. The dissection is then carried upward to include the fourth, third, and second lumbar sympathetic ganglia. Undue traction should not be exerted on any of the tissues handled, especially on the mesentery leading to the sigmoid and other portion of the colon, so as to avoid the possibility of rupture or thrombosis of arteries or branches of arteries which supply the large bowel.

The approach to the lumbar sympathetic ganglia on the right is similar to that on the left, except that the peritoneal incision is made just lateral to the right lateral border of the abdominal vena cava and is carried downward over the right common iliac vein into the true pelvis, upward and mesially along the root of the mesentery of the small intestine, partly across the vena cava for a distance of 15 cm. from the brim of the pelvis, and downward into the pelvis for a distance of 5 to 7 cm. The cecum, small intestine, and ureter are retracted outward and upward. The vena cava is retracted mesially, and the common iliac vein, downward and mesially. In the posterior wall of the peritoneum, just above the brim of the pelvis on the right side, several small veins may be encountered which can be divided and ligated. Further exposure, and removal of lumbar sympathetic ganglia and division of all of the rami and the sympathetic trunk are similar to the procedures employed on the left side. However, the fourth lumbar sympathetic ganglion on the right side usually lies underneath the intravertebral vein and not superficial to it, as it does on the left side.

Presacral Resection With Removal of Second, Third, and Fourth Lumbar Ganglia.—When this procedure is combined with bilateral lumbar trunk resection, the superior hypogastric plexus is readily exposed by extending the lower end of the right peritoneal incision diagonally into the pelvis across the promontory of the sacrum. If the surgeon chooses, the plexus can be equally as well exposed through a midperitoneal incision

extending downward from the bifurcation of the abdominal aorta into the pelvis over the sacral promontory for a distance of 5 cm. On elevating the peritoneal flaps a triangular mass of nerve bundles and fibrous tissue is exposed between the peritoneum in front and the fifth lumbar vertebra and sacrum behind. This mass is known as the superior hypogastric plexus of Hovelacque and its constituents are referred to as the presacral nerves. These sympathetic nerves are a continuation of the intermesenteric plexus and the postganglionic fibers from the first and second lumbar ganglia. The hypogastric plexus (Elaut) also receives additional fibers from the lower end of the lumbar chains and from the inferior mesenteric nerves in the region of the pelvic mesocolon. This mass of nerve tissue is removed in one piece. The dissection is begun at the apex of the triangle which has its point at the bifurcation of the aorta. The nerve fibers are gently elevated from the aorta, common iliac arteries, and left iliac vein, are incised, and are held by a forceps as a retractor. The dissection is directed downward into the pelvis for 5 cm. and laterally to the base of the triangular mass, which usually measures 3 to 4 cm., when all communications are severed, freeing a triangular mass composed of individual nerves, groups of nerves, and dense fibrous bands. The coccygeal artery in the median line usually needs to be included to effect complete removal of the superior hypogastric plexus. The peritoneal incisions are closed with a running continuous suture of catgut.

Splanchnic Resection With Removal of the First and Second Lumbar Ganglia.—The incision[3] employed and the position of the patient on the operating table for extensive sympathectomy are similar to those employed for exploration of the kidney, except that the incision is made to follow anatomic lines. The line of the incision is similar to that of a hockey stick, with the staff portion placed just lateral to the rectus spinous muscles and the club portion extending obliquely downward and forward over Petit's triangle just above the crest of the ilium. At the upper portion of the wound, after the skin and subareolar tissue have been incised, the oblique fibers of the latissimus dorsi muscle are incised, thus exposing the twelfth rib. As the incision is extended downward along the course of the skin incision, the common aponeurosis of the external and internal oblique muscles, and of the transversalis muscle where it fuses with the lateral reflection of the lumbar fascia, is incised until the fusion extends into Petit's triangle, exposing the capsule surrounding the perinephric fat.

The next step in the operation consists of performing subperiosteal resection of the twelfth rib, care being taken not to injure the pleura; the finger is then introduced into the subdiaphragmatic space above the perinephric fat in order to dissect free the subcostal ligament of the twelfth rib, since division of this will allow upward retraction of the

structures, periosteum of the twelfth rib, intercostal vessels, and nerves, which affords a much better exposure of the splanchnic nerves and lumbar ganglia. A wide, illuminated retractor is then introduced into the wound at right angles to the spinal column. It is used to displace the liver forward, then downward, and at the same time to lift the capsule about the perinephric fat with its contents (the kidney and suprarenal gland) in a similar direction. Since the patient is placed on his side with elevation of the opposite loin with the kidney rest, it is not difficult to displace these structures forward and downward, care always being taken to avoid sudden or jerking movements. The tissues are further protected by introduction of a salt laparotomy sponge, with the retractor placed on the sponge to assure against trauma to the underlying tissues. Gentle dissection with moist cotton-ball sponges reveals, first, the splanchnic trunk composed of major and minor and lesser splanchnic nerves, which are about 2 cm. in length. Dissection is carried mesially until the lateral border of the celiac ganglion is exposed. The resection includes a few millimeters of the ganglion with the splanchnic nerves. The first and second lumbar sympathetic ganglia are exposed in turn and are removed in conjunction with the intervening trunk in order to interrupt fibers that pass through the lower end of the thoracolumbar sympathetic trunk.

After hemostasis has been thoroughly controlled, the liver and perinephric fat capsule are allowed to fall into place.

The kidney rest is then lowered, the muscular fascial planes are approximated with continuous and interrupted sutures of No. 1 chromic catgut. The subareolar tissue is approximated with continuous No. 1 plain catgut. The skin is closed with a continuous locking stitch of dermal or silk sutures. No drainage is employed.

The opposite splanchnic nerve and the first and second lumbar ganglia are resected in about ten days following the first operation.

Postoperative Care.—At the conclusion of the operation, while the patient is anesthetized, the rectum is thoroughly dilated. In the moderately severe cases, spontaneous defecation occurs on the following day and continues daily. In the severe cases, soapsuds enemas, retention oil enemas, and mild cathartics may be required. Occasionally, acetylcholine and eserine (physostigmine) have been required during the first few days after operation. As convalescence continues, the abdominal distention subsides, with reduction in the size of the colon. In roentgen examination of these patients months after the operation, the peristaltic movements of the diseased colon are seen to increase in frequency and force, and again present the normal haustra. The colon probably never returns to normal size, but does empty regularly once or twice a day. The splanchnic operation just described has produced more active peristalsis than the other operations and actually has produced diarrhea during the first

ten days after operation, which makes me believe that it is more effective and is indicated in the severe types of Hirschsprung's disease. The hospital diet is similar to that given to children with similar laparotomy wounds. The average stay in hospital is twenty-eight days, ten of which are spent in bed. On dismissal the patient or the parents are instructed gradually to discontinue the use of cathartics, soapsuds enemas, and finally the oil retention enemas as the patient establishes a regular habit

Fig. 6.—Anterior view, illustrating distention of the abdomen accompanied by cachexia due to Hirschsprung's disease.

of defecation in the morning and evening. In less severe cases cathartics and enemas are discontinued immediately; whereas, in advanced cases from six weeks to six months may be required before daily spontaneous defecation takes place.

CLINICAL RESULTS

In reviewing the series of cases of megacolon in which patients were operated on at the Mayo Clinic, I find that twenty-two patients were operated on for Hirschsprung's disease and eight patients were operated

on for atonic dilated colon. One patient with Hirschsprung's disease and two with atonic dilated colons underwent extensive splanchnic and lumbar resection. The rest underwent one of the limited sympathectomies. The ages of the patients with Hirschsprung's disease at the time of the operation varied from five and a half months to nineteen years. Only two patients were nineteen years of age. Three patients were eight years of age, and the remainder were

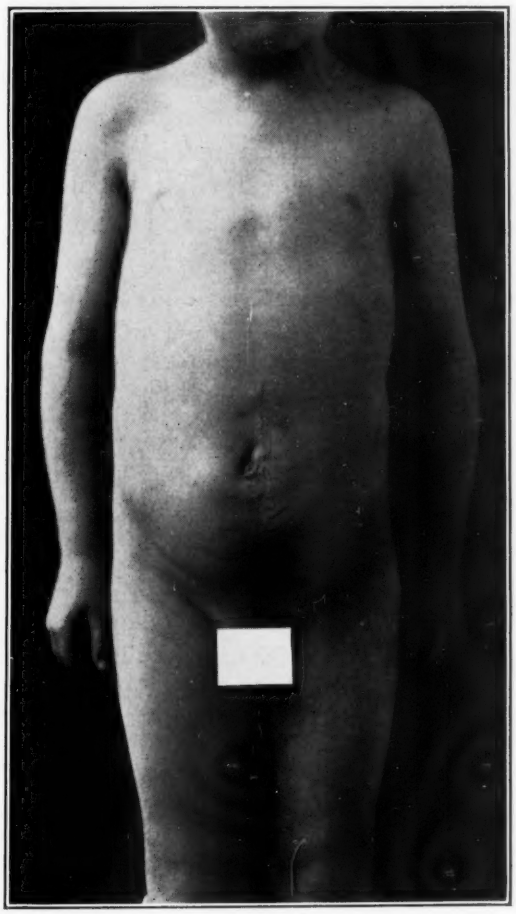

Fig. 7.—Same patient as shown in Fig. 6, four months after bilateral lumbar sympathectomy.

six years of age or younger, two being less than one year of age. In the younger group the disease involved only a limited portion of the colon, with the exception of one patient (G. V.) whose entire colon was involved. The combined operation of bilateral lumbar sympathectomy, with removal of the second, third, and fourth lumbar ganglia and resection of the presacral nerves, failed to give relief in this case. Consequently, this patient was subjected to colectomy, from which he obtained a satisfactory result. The children whose descending colon and sigmoid

were involved obtained excellent results; whereas, those whose disease had extended to include the left half of the transverse colon obtained results from good to fair.

In one case in this group a very limited operation was performed. The patient (H. J.) had involvement of the sigmoid, descending colon and left half of the transverse colon. The operation performed was a left sympathetic lumbar ganglionectomy, the operation of Wade. Immediately following this limited operation the results were satisfactory, but, as time progressed, the patient's symptoms returned and on reexamination I learned that the disease had extended to involve the entire colon. The original operation was performed ten years ago, and in light of the results obtained by bilateral resection of the splanchnic nerves, with inclusion of the upper two lumbar ganglia, it would appear that we are justified in operating again on this child, who is now eleven years of age.

The two patients who were nineteen years of age had involvement of the entire colon. One was submitted to bilateral lumbar sympathectomy combined with resection of the presacral nerves, and the results obtained were only moderately effective. The other was submitted to extensive splanchnic and upper lumbar sympathectomy. The results to date in this case are very satisfactory and much more effective than those obtained in the case of the patient who was submitted to the limited operation which included the lower lumbar and presacral nerves. When I refer to an excellent result, I mean to imply that the patient is able to have spontaneous bowel movements without assistance from cathartics or enemas. A good result implies regular bowel movements with the occasional aid of a mild cathartic, and a fair result implies that the bowel movements are obtained only by daily enemas and mild cathartics.

In most instances the toxic symptoms were relieved. The patient's appetite improved and his languor disappeared. In the less serious cases the distention was relieved and the potlike belly receded, but in those cases in which the entire colon was involved, patients still presented a full belly and peristaltic waves could be detected when the colon became filled with gas (Figs. 6 and 7). These peristaltic waves were usually more active following operation, and postoperative roentgenograms demonstrated definite haustra, while, preoperatively, the roentgenograms presented the characteristic saclike colon. Although the various sympathectomies relieve the symptoms, it is unreasonable to expect the thickened wall of the bowel to resume its normal texture, and consequently most of these patients have a fuller abdomen than normal individuals.

Two patients with Hirschsprung's disease died postoperatively. One died on the operating table as a result of postural influence, since these patients are operated on in the Trendelenburg position. The other died on the day following operation without any pathologic evidence to ex-

plain death other than that the patient was subject to asthmatic attacks which we attempted to control by the administration of epinephrine. A case has been reported in which the patient died three years after an operation which had been followed by excellent results. Death occurred suddenly without explanation; unfortunately, a necropsy was not obtained.

There were eight cases of megacolon of the atonic type, in six of which the patients were submitted to the limited operation which included either bilateral removal of the lower lumbar sympathetic ganglia or resection of the inferior mesenteric nerves and of the presacral nerves, or a combination of two or all three of these operations. The results were unsatisfactory. There were no cures in this group, but in all instances the need for extensive purgation was reduced so that milder cathartics and enemas would initiate evacuation of the bowel. Two patients were submitted to extensive sympathectomy which included bilateral resection of the splanchnic nerves combined with removal of the first and second lumbar ganglia. The postoperative results in both of these cases were excellent, and they have continued to be so to date (a period of nine months). This postoperative period, however, is too short to draw final conclusions. There were no deaths in this group of eight cases.

Sequelae.—No immediate untoward results were observed following the lumbar, intermesenteric, or presacral sympathectomies. Vasodilatation followed in the arterioles of the skin covering the feet, legs and other organs robbed of their sympathetic nerve supply, since the vasomotor fibers are intermingled with the sympathetic motor fibers and naturally are included when the latter are resected. There is a loss of the sweating function over the same area. In women, the sexual organs were not disturbed by sympathetic resection, since I have observed the postoperative effects of resection of presacral nerves in many adult females. Extirpation of the presacral nerve in men does paralyze the mechanism of ejaculation of seminal and prostatic fluid, but does not disturb the libido and potentia. Resection of the presacral nerve does weaken the urinary filling mechanism by decreasing the inhibitory stimuli to the muscles of the bladder and the contracting stimuli of the internal vesical sphincter, but does not produce urinary frequency. The presacral operation has been employed for the treatment of cord bladder. The sequelae of splanchnic resection and removal of upper sympathetic ganglia are similar to the effects obtained by combined bilateral lower lumbar and presacral sympathectomy, with the exception that the vasodilating effects produce increased skin temperatures over a larger area from a level midway between the umbilicus and the symphysis downward over the lower part of the abdomen, including the thighs, legs, and feet. There is loss of the sweating function over the same

area of skin. The potentia and libido are not disturbed in either sex. If there is any change they are both increased, but the ejaculatory powers of the male are lost. This loss of ejaculatory power of prostatic and seminal fluid invariably sterilizes the male, a sequela that adult males should be informed of and which the parents of young males should likewise know.

SUMMARY

In comparing the results reported in literature obtained by operations on the colon with those obtained by operations on the sympathetic nerves, I am convinced that the results of the latter procedures are superior to those formerly obtained by operation on the colon. The mortality is definitely less, and the period of hospitalization is shortened by months.

In selecting suitable cases for operation, typical cases of Hirschsprung's disease should be chosen, in which the response to medical treatment is inadequate. Operation should be performed early in the course of the disease.

On selecting the type of sympathectomy, the degree of the disease should definitely be evaluated and a procedure planned to include sufficient sympathetic fibers to balance the neuromuscular mechanism of filling and emptying of the colon.

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CONGENITAL DUODENAL OBSTRUCTION

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IN 1932, I published a paper on "Congenital Obstruction of the Duodenum in Children,"[1] reporting ten cases, with seven cases successfully operated on. At that time in rather an exhaustive review of the literature, I found numerous cases reported, the first one being cited by Calder in 1752. Most of these articles were reports of autopsy findings. It was not until 1916 that Ernst[2] reported a recovery following an anterior duodenoenteroanastomosis in an eleven-day-old patient with duodenal atresia. Bolling, R. W.,[3] reported a successful result from the operation of duodenojejunostomy in a nine-day-old infant, and Jewesbury and Page,[4] and Rixford[5] reported two cases of extrinsic duodenal obstruction successfully treated. Webb and Wangensteen[6] also collected from the literature six cases successfully treated. Gastroenterostomy was the operation used in this group, but this operation had to be supplemented in one instance by a duodenojejunostomy. Prior to 1932, a moderately careful review of the literature revealed only ten cases of congenital duodenal obstruction that had been successfully operated upon. It seemed at that time that these cases should be more frequently recognized and more often treated successfully, and such has indeed proved to be true. Morton and Jones,[7] in an article devoted to high obstruction in infants, report two cases of intrinsic duodenal obstruction, one of which was relieved by gastroenterostomy only to die twenty months later of pneumonia. The other was relieved by an operation which appears most logical, namely, making a longitudinal incision in the duodenum over the point of obstruction, destroying the septum by electrocautery, and suturing the incision transversely. They also report six cases of extrinsic duodenal obstruction relieved by the same type of operation described by me in 1932. They include three other cases, two of which were successfully operated upon, in which the obstruction was lower in the intestinal tract and are therefore not considered in the scope of this paper. Donovan[8] has reported five cases of intrinsic obstruction relieved by anterior duodenojejunostomy, and Walter E. Lee recently showed a patient at the combined meeting of the Boston

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Surgical Society and the Philadelphia Academy of Surgery who was successfully handled by a jejunostomy for nine months, followed by a posterior gastrojejunostomy.

At the Children's Hospital in Boston, since 1932, there have been an additional twenty-eight cases of congenital duodenal obstruction. There have undoubtedly been other cases, reported or unreported, but these forty-two cases in five years are sufficient to show that this subject is receiving much more attention than it was ten years ago and that the efforts made in treatment are meeting with greater success.

EMBRYOLOGY

The various theories for these anomalies have been discussed in previous papers; suffice it to comment that the intrinsic obstructions are probably due to an arrest in development during the transition which the bowel goes through from a solid cord to a hollow tube, and that this arrest takes place prior to the twelfth week of fetal life. The extrinsic obstructions are likewise due to arrest in development in the normal return of the midgut from the umbilical cord and in its normal rotation.

SYMPTOMS AND SIGNS

The one symptom which is constantly present in these patients with congenital obstruction of the duodenum, regardless of the type, is vomiting. It is worth while to emphasize that vomiting in the newborn in the absence of cerebral injury or infection should suggest malformation in the alimentary tract. In the cases of atresia, the vomiting starts soon after birth or after the first feeding and contains the food ingested plus, in most instances, bile. In the cases of intrinsic stenosis, the vomiting may not occur until considerably later and may be intermittent in character. In cases of extrinsic obstruction due to faulty rotation, the vomiting may start soon after birth or may not occur for several weeks; and it is quite likely to occur in spells. In the intrinsic atresia cases, the physical examination may reveal a distended epigastrium with visible peristalsis or, if the infant be examined soon after he has vomited, the physical examination may be most striking by the absence of abnormal findings. In the intrinsic duodenal stenosis, one may occasionally see the peristalsis of the duodenum running obliquely downward from right to left. In both the intrinsic atresias and stenosis, one is sometimes struck by the prominence of the epigastrium and the sunken appearance of the hypogastrium. In the extrinsic obstructions due to arrested rotation or due to midgut volvulus, the symptoms may start soon after birth or not appear for months or even years. In no instance in our series have the children

developed normally even though they may have been without symptoms suggesting partial obstruction. In the majority of cases, vomiting started soon after birth and persisted with remissions until such time as it became apparent that the infant was failing to progress or

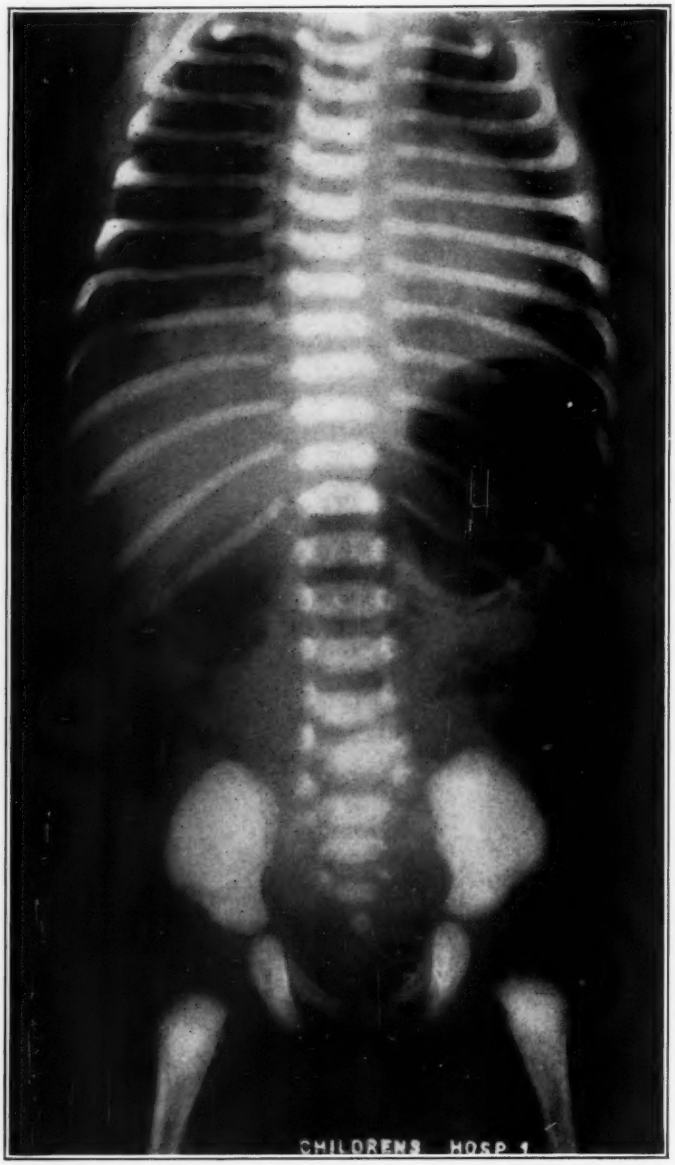

Fig. 1.—(No. 209112) A four-day-old infant with bilious vomiting since birth. Note how clearly outlined the dilated duodenum is without the use of contrast media. Note also small amount of air in lower intestine. Preoperative diagnosis, intrinsic stenosis; postoperative diagnosis, the same.

had become acutely and almost completely obstructed, the variation in the course of the child's progress apparently being dependent on the actual abnormality found. In those patients in whom the obstruc-

tion was caused only by abnormal attachments of the colon or peritoneal folds, the symptoms were milder than in those patients in whom a midgut volvulus had taken place. In the latter situation, the midgut has gone through a twist of 360° or more, which not only almost com-

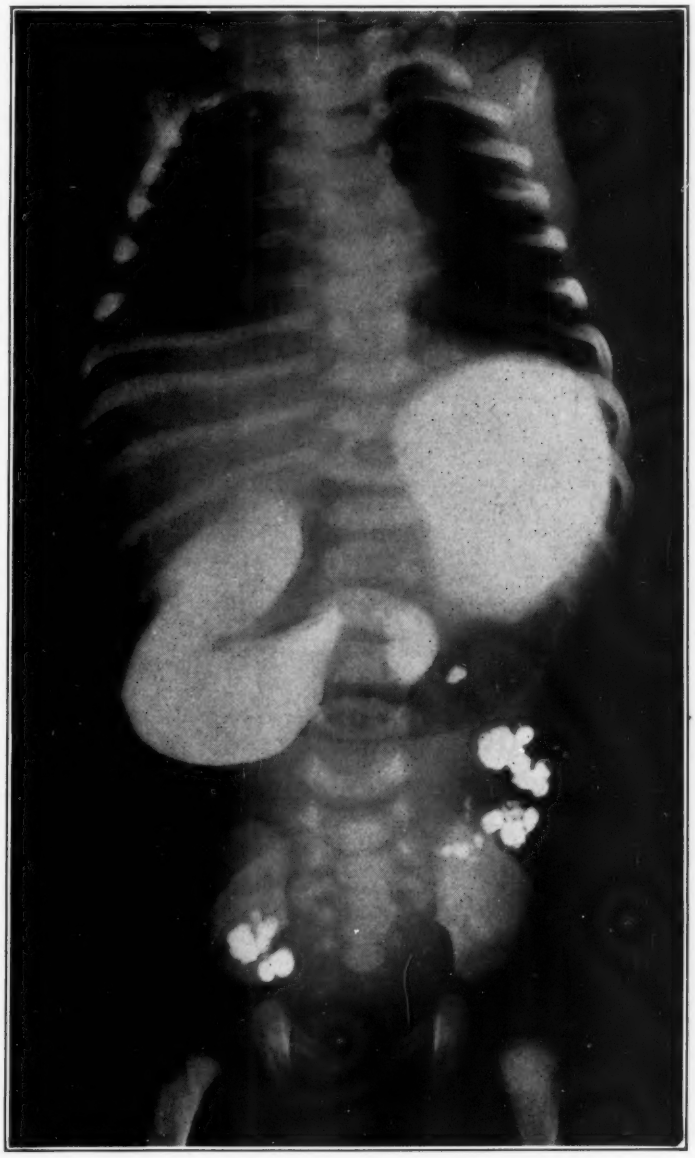

Fig. 2.—(No. 206499) A three-week-old infant with intermittent bilious vomiting since birth. Note dilated duodenum with barium retention. Diagnosis, duodenal obstruction due to malrotation of colon and midgut volvulus.

pletely obstructs the duodenum but also interferes with the circulation of the whole midgut, making an acute situation that demands quick relief. It it interesting to note that in our series when the midgut volvulus has been present, the rotation has taken place, in all but two

instances, in a clockwise direction and that pressure takes place first on the duodenum, obstructing that portion of the intestine and preventing general abdominal or intestinal distention. In both the intrinsic and extrinsic type of obstruction, the x-ray is of great aid in confirming or refining the diagnosis. (Figs. 1 and 2.) On infants under a week of age, it is our practice to first take an x-ray plate without the administration of barium. This will frequently give all the information that is required. When the flat plate shows some air distributed through the intestine or when there is obviously not a complete obstruction, it may be desirable to give barium by mouth and watch its progress by x-ray examination. If there still is doubt as to the type of obstruction, a barium enema may make the diagnosis definite.

The relief of patients with the intrinsic type of obstruction of the duodenum has been undertaken in several different ways, and quite different methods have met with success. Posterior gastrojejunostomy has perhaps been the operation most frequently used and is without question easier to perform than some of the others. This operation has definite disadvantages that were very striking in one of our series. A female infant with intrinsic type of stenosis had a posterior gastroenterostomy performed at the age of six days. The progress was quite satisfactory up to the fifth month of life. At that time the child refused to eat and lost weight. She was placed under excellent pediatric care for two months without improvement. X-ray examination showed that the stoma between the stomach and jejunum functioned very well and that the stomach emptied satisfactorily, but on fluoroscopic examination some of the barium was seen to pass through the pylorus into the duodenum, meet the obstruction, and return by the same route. Examination of the stomach contents showed absence of acid. At the seventh month of life, a posterior duodenojejunostomy was done which was followed by relief and return of normal appetite and development. Webb and Wangensteen have reported a similar experience. Although gastrojejunostomy has met with some success in this series and in other reports, it is not a logical operation for relief of duodenal obstruction. This operation is, theoretically, particularly unsound in the cases of atresia of the duodenum, since in this situation the bile must go back into the stomach, neutralizing the acidity and causing anorexia. Antecolic duodenojejunostomy has been performed successfully by Donovan in five cases. While this operation does not have the drawback of a posterior gastrojejunostomy, it would seem that it might have others. The difficulties are those of having the stoma low enough to adequately drain the duodenum, which is mentioned by Donovan, or the possibility that enlargement or distention of the colon

might cause obstruction either in the colon or jejunum or both. Retrocolic or posterior duodenojejunostomy has been successfully used six times in our clinic and this operation has been successfully used by others. If successful it should provide more adequate drainage for the duodenum and less chance of later obstruction. The drawbacks to this operation are the technical difficulties of its performance. The lack of mobility of the duodenum and the extreme thinness of its wall make an anastomosis in this situation quite difficult. I have recently lost a patient on whom I performed a duodenojejunostomy at the age of five days for intrinsic duodenal stenosis. This infant progressed extremely favorably up to the eighth day then developed peritonitis from which he died. From the postmortem examination it was felt that the peritonitis developed from the anastomosis although no gross leakage was evident. The duodenal wall was extremely thin, and it seemed quite possible that infection might have taken place about some of the stitches. From reports of postmortem examinations there appears to be a moderate amount of uniformity in the thickness of the velum causing these obstructions, and in its situation, between the second and third portions of the duodenum. It would seem that the operation mentioned earlier in this paper as reported by Morton, namely, destruction of the velum causing the obstruction and a plastic on the duodenum to enlarge the lumen, would restore much more normal conditions than any of the other operations so far used. The only possible disadvantage to this operation is the difficulty of approach.

Jejunostomy for feeding has been tried once in our clinic years ago, and it was unsuccessful. It would have no merit in a case with complete obstruction, and it has obvious disadvantages in any case. However, in view of the one successful case shown by Lee, it should not be entirely condemned. In our clinic, the operation of posterior duodenojejunostomy has been the most successful of the operations we have tried both as to immediate and late results. (Table I.)

TABLE I

OPERATIVE TREATMENT OF INTRINSIC OBSTRUCTION OF DUODENUM

OPERATION	NO. CASES	RECOVERIES	DEATHS
Duodenojejunostomy	10	6*	4
Posterior gastroenterostomy	2	1	1
Jejunostomy (for feeding)	1	0	1
Total	13	7	6

*One of these patients had had a posterior gastroenterostomy with subsequent recurrence of symptoms.

The treatment of congenital duodenal obstruction due to arrest in the normal rotation of the colon, whether or not complicated by mid-

gut volvulus, has become quite well standardized at this hospital and with gratifying results. Prior to adopting our present type of procedure, we had attempted to relieve these patients in various ways but with no success. The present procedure consists in making a right paramedian or right rectus incision of adequate length, next delivering the whole midgut and unrotating the volvulus if there is one present. In one or two instances this procedure alone appeared to relieve the obstruction at the time of operation but later proved to be insufficient. The next step in the operation consists in mobilizing the cecum, ascending colon, and proximal half of the transverse colon from right to left until such time as the duodenum is completely visible. If at this time there are any extra folds of peritoneum or bands which impinge on the duodenum and constrict it, they are severed, care being taken, of course, not to injure the superior mesenteric artery. Since adopting this type of operation, there have been twenty-three patients operated upon. Nineteen of these have recovered and have been followed for varying lengths of time from a few weeks up to several years. They have developed normally and without return of abdominal symptoms. Of the four who died, one died of pneumonia three weeks after operation, and one patient, six days old, who had an exfoliating skin lesion, died two days later. At the time of death the skin lesion had become much worse and a diagnosis of pemphigus neonatorum was considered. The other two patients died within two days of operation and might be considered as immediate operative deaths. (Table II.)

TABLE II

OPERATIVE TREATMENT OF EXTRINSIC OBSTRUCTION OF DUODENUM

OPERATION	NO. CASES	RECOVERIES	DEATHS
Anterior gastroenterostomy	1	0	1
Anterior duodenojejunostomy	1	0	1
Reduction of volvulus	1	0	1
Ladd's operation	23	19*	4
Total	26	19	7

*Two of these patients had had previous reduction of volvulus with recurrence of symptoms.

CONCLUSIONS

It seems fair to conclude that if the obstetrician and the pediatrician will regard vomiting of the newborn infant as a signpost demanding investigation of the alimentary tract, many infants may be given a chance for life which was formerly denied them. Posterior duodenojejunostomy is the operation which has proved most successful in our hands to relieve intrinsic duodenal obstruction. Other types of opera-

tion have been successful in other clinics. Of these, the one that seems most logical is the plastic operation on the duodenum described by Morton.

For the extrinsic type of duodenal obstruction it seems clear that the transposing operation described in this paper and in earlier communications is the operation of choice. Reduction of volvulus alone is not sufficient to permanently relieve the obstruction of the duodenum due to malrotation of the midgut.

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THE GENERAL PATTERN AND LOCATION OF THE SMALL INTESTINAL COILS

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DETERMINING the level in the small intestine at which a pathologic process exists is facilitated by the fairly constant anatomic situation and grouping of the small intestine and the characteristic appearance of its various major divisions. The differentiation of the duodenum and jejunum above and the ileum below is easily made by the presence of numerous high mucosal folds in the duodenum and jejunum, inasmuch as these characteristic folds are not obliterated even by marked distention of the intestine. It is possible to make five differentiations in the jejunum and ileum because of the characteristic grouping and situation of their coils in the abdomen.

The arrangement of the small intestine into six primary groups of coils is recognized in both the embryo and the adult. C. M. Jackson in his section on the digestive system in *Morris' Anatomy*, eighth edition, states:

"Even in an embryo of 18 mm., while the intestine is still in the umbilical celom, Mall described six primary coils of the small intestine which could still be recognized after the return of the intestines to the general body cavity, and could usually be identified even in the adult (Fig. 942). In the adult, as also through the various stages of development, loop one forms the duodenum. From the primary groups of coils marked 2 and 3 are developed the greater part of the jejunum, arranged in two distinct groups of loops, situated in the left hypochondriac region. The part of the intestine developed from group 4 of the primary coils passes across the umbilical region to the right upper part of the abdomen. That part developed from group 5 of the primary coils recrosses the median line to the left iliac fossa, while that part derived from group 6 of the primary coils is formed in the false pelvis and the lower part of the abdominal cavity between the psoas muscles. They present what may be regarded as the normal arrangement of the small intestine, having been found 21 times in 41 cadavers examined. Variations from this arrangement occur; the great majority of such variations are, however, not of sufficient importance to require special mention."

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The groups of coils developing from these six primary coils can be recognized roentgenographically when the intestine is filled with an opaque medium, such as a water suspension of barium sulphate, or after moderate distention with air which usually occurs when the intestine is obstructed. The first coil forms the duodenum. The second, third, fourth, fifth, and sixth groups of the coils are the mesenteric small intestine.

Duodenum.—We consider that the duodenum begins at the first crescentic or annular fold distal to the pyloric valve and extends to the duodenojejunal flexure. L. G. Cole has frequently called attention to the fact that the short segment of intestine between the pyloric valve and the first valvula connivens should be considered as part of the stomach or as a separate part of the intestinal tract and not as part of the duodenum. He gave the name of cap to this segment. It is developed from the foregut as is the stomach, while the duodenum and mesenteric small intestine are developed from the midgut.

The duodenum is relatively fixed in position in the form of a loop, the opening of the loop facing to the left and superiorly. The concavity of the loop encircles the head of the pancreas and usually corresponds in size to the head of the pancreas. There are several normal shapes of the loop of the duodenum. The duodenum may be shaped like the capital letters C, V, and U or may be almost rectangular, depending upon the relative position and angulation of the superior, descending, and inferior parts. Due to its relative fixation in a retroperitoneal position, the general form of the loop of the duodenum remains unchanged and is readily recognizable even when markedly distended with air or fluid.

Duodenojejunal Flexure.—This is, as a rule, the highest fixed point of the small intestine. The inferior duodenum usually ascends to the flexure. The flexure is suspended by the fibromuscular ligament of Treitz which passes down behind the pancreas from the lumbar part of the diaphragm. The intestine is only occasionally held to a point at the duodenojejunal flexure. Usually the intestine makes a moderate curve on itself, and less commonly the curve is very wide. The curve at the flexure is usually from posterior to anterior, and in many individuals the angle of the curve can be appreciated only when it is viewed in the oblique or lateral direction. Occasionally the intestine a short distance distal to the flexure is caught up at a second point of fixation, forming a short loop of intestine.

Mesenteric Small Intestine.—The mesenteric small intestine is composed of the second, third, fourth, fifth, and sixth groups of coils of the small intestine. The second group of coils is formed by the intestine as it leaves the duodenojejunal flexure, and the coils lie in the left hypochondriac region. At the distal end of this group, the in-

testine loops toward or beyond the median line to the region of the root of the mesentery and then back to the left to form the third group of coils.

The third group of coils lies in the left lumbar region and may descend into the left iliac region. After forming this group of coils, the intestine loops to the right of the median line in the region of the root of the mesentery and then forms the fourth group of coils. The

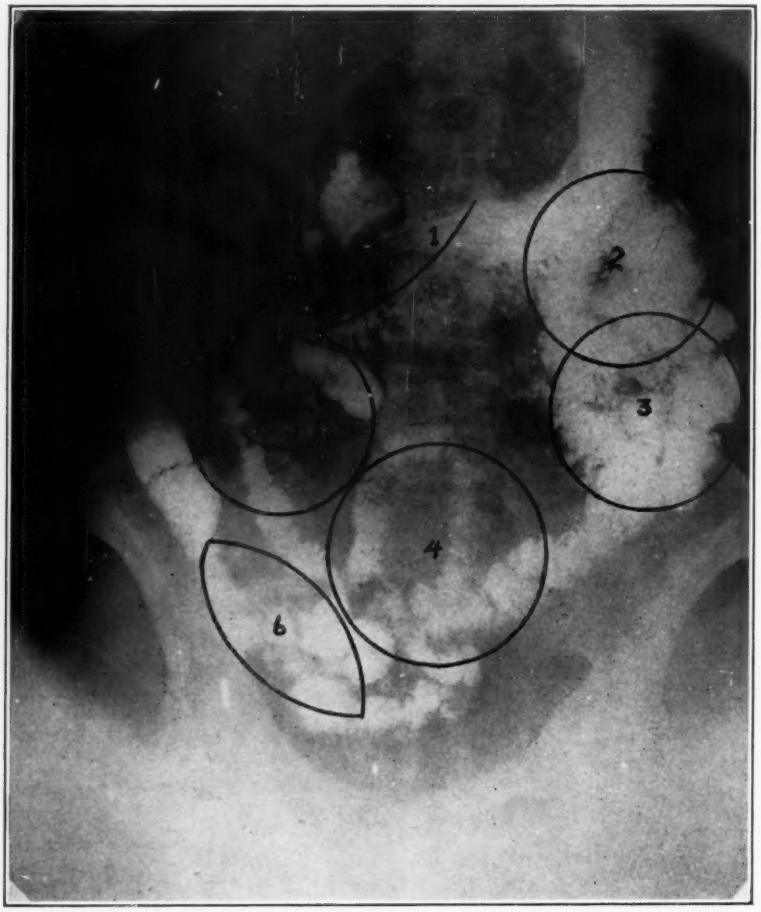

Fig. 1.—One hour after a barium meal, making visible the six groups of coils, stomach, and cecum.

various loops of the second and third groups are quite discrete. Only when they are confined within a relatively small area are they massed together so that the individual loops cannot be recognized.

The fourth group of coils is usually observed in the umbilical and upper hypogastric regions but may extend into the right hypochondrium. The fifth group of coils is usually found in the right lumbar region extending down into the right iliac region. The sixth group of coils is usually located in the lower hypogastric region and pelvis,

the terminal loop ascending from the pelvis into the right iliac fossa to enter the left side of the cecum. The situation of the fourth and fifth groups of coils as observed roentgenographically is somewhat at variance with the description given above by Jackson.

Distinct visualization of the fourth, fifth, and sixth groups largely depends upon the build of the individual. In hypersthenic individuals most of the loops are out of the pelvis, the liver is high, and the loops can be individually recognized. In asthenic types of individuals the

Fig. 2.—One-half hour after the barium meal, showing barium in the stomach and the first five groups of coils. Figs. 2, 3, and 4 show the same case.

liver is relatively low in position and the loops of the lower groups of coils may be so massed together in the lower part of the abdomen that it is impossible to recognize their outlines. Upward displacement of the intestines by a filled urinary bladder also tends to mass the loops together.

There is also a characteristic arrangement of the intestinal loops within the groups of coils. In the second and third groups of coils the individual loops lie transversely in the abdomen, and in the fourth,

fifth, and sixth groups the individual loops lie vertically. Between each group of coils a relatively longer loop of small intestine extends back to the root of the mesentery. This formation is most characteristically seen between the second and third groups of coils.

Displacement of the first and second groups of coils into the right hypochondrium and lumbar region is the rule in association with incomplete rotation of the colon. Rarely it occurs as an isolated abnormality. In both instances, the duodenojejunal flexure may be displaced

Fig. 3.—One and one-half hours after meal. The head of the meal is at the splenic flexure. The distended bladder displaces the fourth and sixth groups upward.

out of its usual position. Another unusual anomaly is to have the upper loops of the mesenteric small intestine festooned across the upper abdomen.

Motor Phenomenon of Small Intestine.—Food or other substances in the small intestine are propelled through the gut by peristaltic contractions which may develop at any level and progress analward. The most proximal wave begins in the middle of the cap, partially emptying the cap, and progresses through the duodenum to varying levels in the mesenteric small intestine. The first bolus of barium evacuated from the cap may be carried as far as the fourth group of coils before

the peristaltic contraction disappears. Under normal conditions the rate of progress of the peristaltic contraction is more rapid in the upper part of the small intestine. In the duodenum and second, third, and fourth groups of coils it progresses at the rate of two to four centimeters per second. In the lower part of the small intestine the wave of peristalsis moves much slower, usually less than one centimeter per second. Under normal conditions of irritability the longitudinal length of a peristaltic contraction wave is approximately one centimeter. With abnormal irritability, the longitudinal length of the peristaltic wave is increased as is also its rate of progress.

Fig. 4.—Two and one-half hours after meal. Bladder empty, allowing the coils to drop down.

Progressive peristaltic contractions are frequently preceded by stationary tonic contraction rings. As a tonic contraction becomes a progressive peristaltic contraction, there may be many abortive attempts at progression. During fluoroscopic examination of the small intestine we have never observed to-and-fro movements of the barium. The nearest approach to this phenomenon has been the change in the barium shadow produced by these abortive attempts at progression in tonic contraction rings situated fairly close together in the intestinal muscle.

Caliber of the Lumen of the Small Intestine.—In the living subject the intestine is normally in a state of tonic contraction; it is contracted on itself or its contents. For this reason the caliber of the lumen of any section of the small intestine will depend upon the degree of filling or distention. Certain normal variations in the rapidity of emptying of the stomach and the rapidity of passage of the meal through the small intestine will, therefore, modify the distribution of the meal and the pattern of the intestine. Rapid uniform emptying of the stomach will give a large caliber to the intestine throughout

Fig. 5.—Atypical arrangement of the groups, the third and fourth groups being to the right and the fifth to the left of the median line. The second group is slightly to the right of its normal position.

the entire length in which the meal is present. With slow or moderately rapid emptying of the stomach, the caliber of the lumen of the intestine will be small or moderate if the passage of the meal through the intestine keeps pace with the emptying of the stomach. If the passage of the meal through the intestine is more rapid than the entrance of the meal into the intestine, the caliber will be small, but usually uniform. If the stomach empties itself intermittently, the distribution of the meal and the caliber of the small intestine will be irregular.

Pattern Produced by Obstruction of Small Intestine.—The root of the mesentery of the small intestine runs obliquely downward from the region of the left upper quadrant and across the midline to the right lower quadrant, a distance of six to eight inches, and is attached to the posterior abdominal wall. The anterior border of the mesentery is attached to the small intestine and is necessarily as long as the gut, about twenty-two feet. Due to the great length of the small intestine

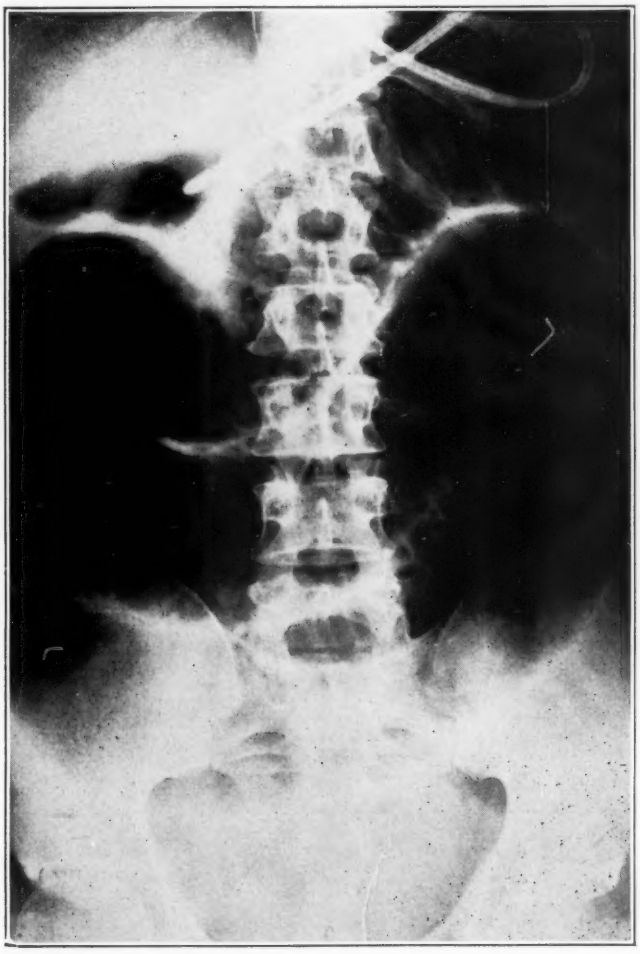

Fig. 6.—Pattern produced by marked distention of the second, third, and fourth groups of coils due to obstruction in the upper ileum. In spite of the marked distention, the markings of the primary mucosal folds are retained.

and the limiting space of the abdominal cavity, the mesentery is arranged in a folded or frilled fan-shaped manner; the small intestine itself is in a collapsed or slightly distended state and arranged in groups of coils as described above. The second and third groups of coils of the small intestine lie usually to the left of the root of the mesentery; the fourth group lies on both sides; the fifth lies to the right, and the sixth to the left with some overlapping to the right.

The individual coils in the groups are produced by the varying length of the mesenteric attachment, the ends of the coils having a short mesentery and the looping portion having a progressively longer attachment. These coils normally fold on themselves to form short loops, although this folding pattern does not remain fixed.

The pattern produced on the roentgenogram in any case of obstruction of the small intestine is determined by the following factors: (1) Site of obstruction, (2) degree of gas distention, (3) length of gut distended, (4) the closed abdominal space.

When an obstruction occurs at any point in the small intestine, the gut proximal to this area becomes gradually distended with air, the amount of distention depending upon the degree of obstruction, the elapsed time, and the amount of air swallowed by the patient. We have always been under the impression that distention of the small intestine due to obstruction causes an elongation of the gut as well as an increase in the diameter. Sperling and Wangensteen,[1] however, in producing acute obstruction in dogs, noted a definite shortening of the bowel which in some instances amounted to as much as 33 per cent. These findings are directly the reverse of what happens in distention of the stomach and colon; here we can easily visualize an increase in length as well as diameter by means of the roentgen examination. The distention of the small intestine, when marked, causes an obliteration of all but the permanent mucosal folds, the latter causing a constriction of the wall which results in a characteristic pattern that can be likened to stacked, miniature balloon tires. Occasionally in cases of chronic obstruction of the small intestine, even these primary mucosal folds may be wiped out. With distention the individual coils unfold and are displaced out of their relatively normal positions by the internal pressure. Thus we may see a continuous coil of distended gut passing across the entire abdomen turn on itself and swing back toward the root of the mesentery, from where another coil may swing away in a different direction, the ends of the coils being held near the root of the mesentery by their shorter attachments. Occasionally a case is seen in which a relatively short length of small intestine, ten to twelve inches, is distended with air; these short loops are always curved, indicating the influence of the mesentery on the pattern produced.

The distended coils of the second group tend to assume a horizontal position across the abdomen, usually not extending very far beyond the midline to the right; the coils of the third group extend horizontally across the abdomen. The distended coils of the fourth, fifth, and sixth groups tend to assume an oblique and vertical position in the abdomen.

In a case showing marked distention of the bowel, it may be impossible to distinguish distended colon from distended small intestine on the roentgenogram. It is then advisable to administer a barium

enema to determine the presence or absence of obstruction in the colon. A diagnosis of an obstructing lesion in the colon distal to the cecum is important information to the surgeon in determining his operative procedure. In no case of complete obstruction should a barium meal be given by mouth as the impaction of the barium that results will create an added hazard to both the surgeon and patient. If one decides that barium by mouth may give additional important findings, small amounts of a thin watery mixture may be used, and then only in cases of partial obstruction.

The differential diagnosis between complete mechanical obstruction of the colon, paralytic ileus, and mesenteric thrombosis is often extremely difficult when there is marked distention of both the large and small bowel in these conditions. The barium enema examination usually detects the presence of a mechanical obstruction. Visualization of the entire colon by means of the barium enema in the absence of an obstructing lesion limits the diagnosis to a paralytic ileus or mesenteric thrombosis, an impossible differential diagnosis by means of the roentgen examination. However, early in mesenteric thrombosis there is no distention of the gut with air, and when a barium meal is given by mouth, the head of the column stops in the second or third group of coils, the stomach retaining most of the meal. Practically no change will occur in the progress of the meal for as long as forty-eight hours or until vomiting and gas distention of the bowel occur. The diagnosis of mesenteric thrombosis is very rarely made before gas distention of the gut is present, and even then a positive differential diagnosis is usually impossible.

Large amounts of fluid or semifluid fecal material in the distended bowel may and usually do give an atypical pattern on the roentgenogram; large collections of gas may not be present, occurring only as numerous small bubbles quite uniformly mixed with the fluid. In such cases, the contrast between gas-distended bowel and surrounding soft tissue shadow is considerably diminished due to the density of the fluid. If the roentgenogram is made in the upright posture, fluid levels will be seen in the loops of distended gut.

It is sometimes possible to determine quite accurately at which level in the small intestine an obstruction exists, but usually this is difficult to do with any degree of certainty, particularly when the obstruction is low with many distended and superimposed coils filling the abdominal cavity. However, the roentgenologist can frequently direct the surgeon where to make his incision for the nearest approach to the obstructing lesion.

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FUNDAMENTAL CONSIDERATIONS IN THE OPERATIVE TREATMENT OF ADVANCED INTESTINAL OBSTRUCTION WITH ESPECIAL REFERENCE TO THE MANAGEMENT OF CASES COMPLICATED BY GANGRENE OF THE INTESTINE

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TREATMENT of bowel obstruction should be planned to cure the bodily injuries which obstruction produces. I begin, therefore, with a discussion of the nature and extent of these injuries. This, I hope, will justify in the mind of the reader the treatment I advocate. Authorities on bowel obstruction seem now to be in substantial agreement in regard to the chief effects thereof as given in the following summary.

SYSTEMIC EFFECTS OF OBSTRUCTION

Dehydration and the starvation always associated with it are the chief causes of death following untreated obstruction.

Obstruction of the small intestine causes a prodigious loss of water and electrolytes by the blood. This dehydration is nearly always accompanied by an alkalosis. It accounts for the rapid, feeble pulse and low blood pressure observed in patients with ileus. It accounts also for the failure of the kidneys to excrete nitrogenous wastes. The increased viscosity of the blood may cause obstruction of the intrinsic arteries of the heart if these are already narrowed by sclerosis. It makes weaker the already enfeebled circulation through the bowel wall. It deranges the entire metabolism.

We shall discuss later the possibility that systemic effects are under certain conditions produced by toxins absorbed from the obstructed bowel.

EFFECT OF OBSTRUCTION ON THE BOWEL

These on final analysis are due to interference with circulation, caused, (1) by distention, (2) by direct pressure on the bowel, and (3) by obstruction of the blood vessels of the mesentery.

Effects of Distention on the Bowel.—As the intraintestinal pressure increases, the quantity of blood flowing through the intestinal wall decreases. The flow ceases entirely when the intraintestinal pressure equals the systolic blood pressure. Distention alone can cause necrosis of the bowel wall. The bowel, however, endures the anemia caused by distention astonishingly well. The writer has demonstrated that

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detached pieces of rabbit intestine remain alive in Ringer's solution for twelve to fifteen hours. Under clinical conditions the bowel probably remains viable much longer than this when distention is the sole impediment to its circulation. The pressure within it will take a long time to reach a height sufficient to cut down the blood flow to a dangerous extent; furthermore, regurgitation of its contents will keep down the pressure. It is to be noted that prolonged distention, even though it may not injure the bowel beyond possibility of recovery, may conceivably damage the mucosa enough to make it permeable to toxins.

While the large bowel can resist the effects of distention for a longer time than the small bowel, it is much more liable to ultimate injury thereby. This is because the colon cannot readily empty its contents backward into the small intestine. The muscular coat of the bowel loses the power to contract when stretched beyond a certain extent. When this occurs the bowel can no longer protect itself against distention by evacuation of its contents. A bowel which has been overdistended for some time regains but slowly the power to contract. For this reason a short-circuiting operation or even enterostomy may not relieve obstruction for several days.

Effects of Direct Pressure on Bowel Wall.—Direct pressure upon the bowel wall by an impacted foreign body, e.g., by a gallstone or by the neck of a hernia or a peritoneal band, may cause necrosis thereof. The danger of this is increased by intestinal distention. Areas of bowel weakened by this cause are common sites of perforation.

Effects of Interference With the Mesenteric Blood Vessels.—Clinically, this is the most common and dangerous cause of circulatory disturbance in the bowel wall. In the form of interference most frequently encountered, the mesenteric veins are obstructed while the mesenteric arteries are not, for example, incarceration of the bowel in hernia or beneath a peritoneal band. Under these conditions blood flows into the wall of the bowel under the full force of the systolic blood pressure but cannot leave it. We have shown that this subjects the capillaries of the bowel to the full force of the systolic blood pressure. This ruptures the capillaries and rapidly demolishes the entire bowel wall.

ABSORPTION OF TOXINS FROM OBSTRUCTED BOWEL

Before the discovery that intestinal obstruction causes a profound dehydration, death from all forms of intestinal obstruction was attributed to the absorption of poisonous materials from the bowel. This theory dominated treatment until recent years and was the cause of an enormous amount of experimental work, the major portion of which is now of historical interest only. Our present-day view of the intoxication theory, briefly stated, is as follows: The content of ob-

structed bowel is less toxic on intravenous injection than the content of normal bowel. The healthy intestinal mucosa prevents the passage into the blood stream of any toxic material conceivably present either in normal or obstructed bowel. The absolute proof that toxic material is absorbed from the obstructed bowel has never been produced. There are good reasons for believing that the body is protected in a fairly adequate way from such absorption. Two of these are the following:

1. When the mesenteric vessels are obstructed, nothing but transperitoneal absorption can take place. This is probably slight, because patients often live for days with pieces of gangrenous intestine without showing noteworthy signs of intoxication. The peritoneum can isolate a loop of gangrenous bowel as readily as it can an abscess.

2. Distention itself, if due to an intraintestinal pressure higher than the diastolic blood pressure, prevents all absorption from the bowel by way of its mesenteric vessels, even though the mucosa be damaged. Since untreated bowel obstruction is accompanied by a marked fall in the blood pressure, it is probable that the intraintestinal pressure in any damaged loop of bowel will be as high as the diastolic blood pressure and that, therefore, no absorption from it can take place.

Absorption of toxins can occur only in the presence of a devitalized intestinal mucosa and an adequate circulation through the bowel wall. These conditions are present when the intestinal mucosa has been partially devitalized by prolonged and great distention which is rather suddenly relieved. They might be present also after a low blood pressure had been elevated by the administration parenterally of a considerable quantity of water and electrolytes. This elevation of blood pressure, together with the decrease in viscosity of the blood which accompanies it, can conceivably restore the flow of blood through a distended loop of intestine, which before had been almost without circulation, and which, because of this fact, had a devitalized mucosa.

Most surgeons have observed patients collapse or die with symptoms suggestive of profound intoxication after the abrupt relief of a bowel obstruction. The writer has never observed an outcome of this kind following the administration of considerable quantities of fluid, but the possibility of its occurrence in advanced cases of ileus is worthy of thought.

TREATMENT

Diagnosis is outside the scope of this paper. Suffice it to say that before operative treatment is undertaken, the presence, site, and stage, whether early or late, of the obstruction should be determined.

We are of the opinion that the appalling death rate following operation for bowel obstruction has been in great part due to the idea that the diagnosis is an indication for immediate operation. The con-

trary is true in all but early cases. The diagnosis of advanced ileus is a contraindication to operation until the patient has been prepared therefor. This preparation consists essentially (1) in the relief of starvation, dehydration, and alkalosis by the parenteral administration of adequate amounts of salt solution and glucose and (2) in the relief of gastric and intestinal distention. The second of these requires extended comment.

When the obstruction is in the large bowel beyond the cecum and is complete, it is our practice to relieve it by a cecostomy, performed through a McBurney incision by amputating the appendix and dilating the opening through the cecal wall until it will admit a good-sized tube. This is then inserted to a length of several inches and the end of the cecum is invaginated around it on the principle of a Senn gastrostomy by two or three superimposed purse-string sutures of catgut. The tube is pushed through a flap of omentum which is tucked around the cecum, and the peritoneum is tightly closed about it. The other layers of the abdominal wall are very loosely sutured. We have been able nearly always to empty the colon in this way and have observed little or no fecal drainage after removal of the tube. It may require a week or longer to empty the colon and to give it time to regain a healthy tone. Then resection can be done with comparative safety and a death rate which should not exceed 5 per cent. Resection at an earlier time is difficult and dangerous. Between cecostomy and resection the patient should be given a full, low-residue diet.

If the obstruction is situated anywhere from cecum to pylorus, we now relieve distention by continuous gastric lavage. We formerly relied on a Witzel jejunostomy for this purpose, but we have found the lavage equally efficient except for very advanced cases. It takes the courage of conviction to persist in the attempt to deflate the bowel for perhaps several days. Our clinical experience, however, has now been sufficient to justify this treatment. It must be remembered that an overdistended bowel regains very slowly the power to contract; also, that it can be trusted to retain its viability for a long time. The slow relief of the distention is a safeguard against an overwhelming absorption of toxic material. Several of our patients, apparently in a hopeless condition on admission, were cured by the treatment just described, without any operation at all. In these it is possible that the obstruction was due to a kinking of the bowel over a peritoneal band and that the relief of the distention undid the kink. Patients so relieved should, of course, be kept under close observation for a long time.

How long should the attempt to deflate the small bowel be kept up? In general, the more slowly deflation is accomplished, the more neces-

sary it is. It may be completed in a few hours in early cases. In advanced cases, it may be impossible to deflate completely the lower reaches of the obstructed bowel. Sometimes placing the patient on his side or in the prone position may be a great aid to deflation. It is safe to operate when abdominal distention has been greatly reduced, when borborygmi can be heard, and when the general condition of the patient is good.

If the site of the obstruction has been determined with fair accuracy, the operator can make the abdominal incision which will permit the best possible exposure of the lesion. It is needless to dwell upon the difficulties and dangers of this exposure in the presence of greatly distended loops of bowel. If there is no gangrene of the intestine, simple relief of the obstruction is all that is required. If the contrary is true, we believe that gun-barrel enterostomy following removal of the gangrenous piece of bowel is the best treatment. We never perform intestinal anastomosis at this time. The patient, despite all efforts to improve his condition, is still a poor risk for operation; the overstretched bowel is in no condition for suturing, which is followed too often by death.

The fear of leaving a gangrenous piece of bowel in the abdomen is, I believe, the reason which prompts many surgeons to operate upon patients with advanced ileus as soon as possible. My experience has led me to believe that, even if this formidable complication is suspected, it is safer to defer operation until the preparations described above have been made. As already stated, the peritoneum can wall off a piece of gangrenous bowel as readily as it can an abscess, and the one may be attended by no more systemic disturbance than the other. I submit case reports of three consecutive cases to enforce this point.

Case 1.—White woman, aged thirty-eight years. Admitted June 23, 1936. Discharged August 6, 1936. Complaint: severe abdominal pain, nausea, and vomiting. Onset June 21, sudden, while on a 200-mile auto trip; required morphine.

In March, 1934, the patient had had a double salpingectomy, Gilliam suspension, and removal of a small cyst from one ovary. A year before admission she had had a short attack similar to the present one.

The patient was acutely ill, in extreme dehydration. The abdomen was distended, and there was marked tenderness and muscular defense above the pubis. No bowel sounds were heard, but some gas had been expelled with an enema. The uterus was of normal size and in normal position. Behind it the pelvis was filled by a tense boggy mass.

Temperature, 101°; pulse, 120; respirations, 24; white blood count, 14,050 with 94 per cent polymorphonuclear cells. The urine contained albumin, casts, and red blood cells. X-ray examination revealed a ladder pattern of distended loops of small intestine.

Treatment consisted of morphia, continuous gastric lavage, and intravenous injections of salt solution and glucose. The abdominal distention became markedly reduced and bowel sounds could be heard. The temperature remained around 101°.

Operation: June 26, the abdomen was opened through a suprapubic incision. A greatly distended, totally gangrenous loop of bowel was found in the pelvis. It had been strangulated by passing around the right round ligament between the uterus and the anterior abdominal wall. It was ruptured while being removed, but its escaped contents were quickly and completely removed from the pelvis by aspiration. A gun-barrel enterostomy was done. Omentum was disposed around the emerging ends of bowel. Penrose drains without gauze were inserted into the bottom of the pelvis and brought out below the enterostomy. The incision was closed in layers with a continuous suture of plain catgut for the peritoneum and loosely applied chromic catgut sutures for the anterior sheaths of the recti. The skin was partially closed with interrupted sutures of silk.

Postoperative course was relatively uneventful. The treatment for about three days was the same as before operation, except that water was given through the distal end of the enterostomy instead of by vein. Then the Levine tube was removed and soft food and fluids given by mouth. There was no excoriation of the wound, which was kept clean by manually applied suction. Because of the generally poor state of the patient, closure of the enterostomy was deferred until July 22—twenty-six days—when her condition was excellent.

The wound was reopened, the bowel ends freed, and a lateral anastomosis done. The abdomen was closed completely except for a Penrose drain inserted down to the posterior surface of the uterus and carefully protected from the site of the anastomosis.

It is noteworthy that the wound healed almost by primary union, although three weeks after operation, when the patient was at home, she developed a small abscess at the lower end of the incision. She has since remained in good health.

CASE 2.—White man, aged twenty-two years. Admitted on January 29, 1937. Discharged March 3, 1937. Complaint: severe, crampy pain in the epigastrium. Vomiting began ten hours after onset, never fecal. Passed a little gas and some solid material with enema.

He had had appendectomy in August, 1936. Health had been good until onset of present illness.

Seen in consultation on February 2. General condition good except for moderate dehydration. Abdomen was somewhat distended; bowel sounds present. A very tender sausage-shaped mass felt above and parallel to Poupart's ligament; tense mass felt by rectum in right side of pelvis.

White blood count 27,500, with 92 per cent granulocytes. Urine negative. X-ray plate of abdomen showed normal colon and a ladder pattern of distended loops of small bowel.

Diagnosis: obstruction of small bowel with gangrenous loop. Continuous gastric lavage was started and 2,000 c.c. of normal salt solution with 5 per cent glucose given by vein.

Operation: (Dr. John Owen.) February 2, 1937. A gangrenous loop of terminal ileum 40 cm. long, obstructed by a band due to the appendectomy, was removed and a gun-barrel enterostomy done. Abdominal wall was closed as described for Case 1.

Postoperative course entirely uneventful. No wound complication. On February 26, 1937, the distal end of the enterostomy was invaginated into the cecum, and a lateral anastomosis done between the proximal end of the ileum and the ascending colon. Recovery was uneventful.

CASE 3.—White boy, aged fifteen years. Admitted January 12, 1933. Dismissed March 5, 1933.

Onset five days before admission with abdominal cramps and vomiting. Vomitus bile-tinged for three days, never fecal. General abdominal soreness since onset. Dark blood in stools for last two days.

Past history uninforming except that patient had had "stomach upsets" at intervals since birth.

Physical examination showed the boy to be in good general condition except for dehydration. Abdominal distention was not great. There was marked muscular defense and tenderness over the McBurney region. A mass could be felt by rectum in the right side of the pelvis.

Temperature, 100; pulse, 106; respirations, 18; white blood count, 19,950 with 80 per cent granulocytes. Urine negative.

A diagnosis of appendical abscess with associated bowel obstruction was made. After a short period of gastric lavage and the administration of 1,000 c.c. of salt solution by vein, operation was performed.

A McBurney incision was made, whereupon the true nature of the trouble was discovered. A gangrenous piece of bowel was found in the pelvis. The McBurney incision was closed, and a suprapubic incision made. The gangrenous piece of bowel was resected. It proved to be a double intussusception of the ileum of uncertain origin. A gun-barrel enterostomy was established and the wound closed as described for Case 1.

The obstruction had evidently been high, because there was great excoriation of the skin around the enterostomy. Nourishment and water were given through the distal limb of the enterostomy. Continuity of the bowel was restored by lateral anastomosis on February 16, 1933. Recovery was then uneventful with good healing of the wound. The boy has remained well.

COMMENT

The interval between operations could have been shortened with safety in all of these cases. Its length will depend in any given case upon the general condition of the patient and the time required for intraabdominal or wound abscesses to heal. The bowel, atonic because of prolonged overdistention, may not resume a normal tone for several days after enterostomy. During this time the patient's nutrition cannot be improved. The second operation for these reasons cannot usually be done sooner than two weeks after the first.

Our experience with these patients demonstrates that gangrene of the bowel may cause amazingly little intraabdominal or systemic disturbance; that the fear of its presence should not lead to operation before the patient has been prepared therefor; that with proper preoperative preparation followed by excision of the gangrenous bowel and enterostomy, this serious complication of bowel obstruction can be successfully treated.

HERNIA AS AN ETIOLOGIC FACTOR IN ACUTE INTESTINAL OBSTRUCTION

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HERNIA provides potentially those anatomic and mechanical conditions which are generally recognized as essential to the production of intestinal obstruction, and it is not extraordinary to find that the therapeutic methods of the preantiseptic period made little progress in limiting the incidence of this complication. It was, however, to be anticipated that the advent of asepsis, inaugurating the modern era of surgery and establishing the efficiency of the open methods of treatment, might promise a popularization of these methods and, in consequence, prognosticate a prophylactic reduction of this dreaded obstructive complication. That this promise has to some degree been realized is amply attested by the fact that open operation for radical cure of hernia is today one of the most commonly performed procedures in the whole field of general surgery. On the other hand and in spite of the widespread attack upon this potential source of major complications, the inescapable fact remains that, relatively and absolutely, hernia at this time continues to head the list of causes of acute intestinal obstruction.

So far as our own material is concerned, the answer to this enigma is to be found not so much in statistical data as in certain sociologic and psychologic factors which must be given due consideration if these data are to be correctly interpreted. For example, the figures constituting the present report are based upon clinical material drawn almost exclusively from a social stratum which is traditionally antagonistic to the principle of prophylaxis and which views surgical therapy as a kind of Pandora's box whose powers are to be invoked only as a last resort. Reference to the records of our series of acute intestinal obstruction due to hernia discloses eloquent evidence of this state of mind which may justly be held accountable for the significant facts that 82.8 per cent of the group were victims of hernia for periods varying from two years to forty years preceding the onset of the final obstructive manifestations; that more than half the cases had been obstructed for twenty-four hours to ten days before seeking treatment; and finally, and in consequence of the foregoing, that the group as

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a whole presented a mortality rate approximating 30 per cent. It may, of course, be said in extenuation that this attitude toward hernia is not confined to any single class or group and that the very chronic nature and frequency of this condition bespeak a familiarity which breeds in general a contempt for its complications. Be this as it may, the fact remains that hernia is a potential source of acute intestinal obstruction, and prophylactic herniotomy is surely a small price to pay for insurance against the possibilities of this catastrophe.

The rôle of hernia as an etiologic factor in acute intestinal obstruction is indicated by an analysis of the records of the Fourth Surgical Division and the Children's Surgical Service, Bellevue Hospital, for the period from January 1, 1924, to January 1, 1937. Among the 52,351 surgical cases admitted during this period, 3,432 were classified as hernias of various types and of these latter 130 were associated with acute intestinal obstruction; whereas, during the same interval, the number of intestinal obstructions from all other causes combined totaled only 109 cases. On the basis of these figures, therefore, we may be permitted to conclude (1) that for this series the incidence of intestinal obstruction due to hernia, external and internal, is approximately 4 per cent and (2) that, while hernia constitutes but 6.5 per cent of general surgical conditions, it is, nevertheless, the inciting factor in 54 per cent of all acute intestinal obstructions.

In view of the fact that certain conditions may simulate but not actually qualify for inclusion in the group of acute intestinal obstruction due to hernia, it is necessary to establish specific criteria upon which to base the selection of cases comprising this group. It must be recognized, for example, that neither irreducibility or incarceration is necessarily synonymous with obstruction, and it is furthermore important to recall that pure omental hernias complicated by strangulation or torsion may, as a result of reflex peritoneal irritation, ileus, vomiting, and pain associated with characteristic local hernial signs, closely approximate the accepted clinical picture of obstruction. In consequence, the selection of cases for admission to the present series has been determined not only by the presence of the classic clinical symptoms of obstruction, but more particularly through the anatomic demonstration within the sac of an intestinal segment whose lumen, regardless of type of mechanism, has been encroached upon to such a degree as to preclude the maintenance of its excretory function.

As thus classified, there were 130 cases of acute intestinal obstruction induced by some type of internal or external hernia, and this number constitutes 3.8 per cent of all hernias admitted during the period of this study. Integration of this group shows that males

TABLE I

ANALYSIS OF 130 CASES OF HERNIA WITH INTESTINAL OBSTRUCTION

TYPE OF HERNIA	NUMBER	SEX		AVERAGE AGE	AVERAGE DURATION OF SYMPTOMS	DEATHS	MORTALITY RATE
		M	F				
Indirect inguinal	79	76	3	39.3	39.1 hr. (71 cases)	12	15.0%
Direct inguinal	7	4	3	52.0	43.4 hr. (6 cases)	1	14.0%
Femoral	26	8	18	55.3	67.2 hr. (25 cases)	15	57.5%
Umbilical	7	0	7	55.2	47.3 hr. (6 cases)	4	57.0%
Ventral	8	1	7	51.0	46.5 hr. (6 cases)	4	50.0%
Interstitial	1	1				1	
Semilunar	1	1				1	
Internal	1	1				0	
Group totals	130	92	38	44.1 (130 cases)		38	29.23% (130 cases)

predominate over females in the ratio of 92 to 38; that the average age for the group as a whole is 44.1 years; and that there were 38 deaths, establishing a mortality rate of 29.23 per cent.

It is apparent from comparative study of the various types comprising this group (Table I) that, while the indirect inguinal variety with 79 cases predominates among the causes of hernial obstruction, it presents at the same time the lowest figures for duration of acute symptoms (39.1 hours) and age of onset (39.3 years), both of which factors undoubtedly have a significant bearing upon the minimum mortality rate of 15 per cent recorded for this type of hernial obstruction. On the other hand, femoral hernia, which is second in importance from the standpoint of frequency, with 26 cases, shows the maximum figure for duration of acute symptoms (67.2 hours), the highest average age (55.3 years), and as a logical consequence, the maximum group mortality rate of 57.5 per cent. Among the less common obstructive hernias, the ventral, umbilical and direct inguinal types correspond very closely in respect to average age, duration of symptoms, and mortality rate, with the single exception that the latter (direct inguinal) is distinguished by its minimum mortality figure of 14 per cent. For the whole group, males preponderate over females in the proportion of 92 to 38, with the indirect inguinal type showing a special predilection for males, the femoral, umbilical, and ventral for females, while the direct inguinal hernias are about equally divided between the two sexes.

There were three instances in which obstruction complicated rare types of hernias. The first, a semilunar or Spiegel's hernia, occurred in a male of fifty-four years of age and presented clinical symptoms of

acute intestinal obstruction without demonstrable evidence of its site. At operation, there was found protruding through the left semilunar line just above Douglas' fold, a small sac containing a loop of small gut, which was constricted at its neck by a ring of posterior sheath through which it passed. The second instance was noted in a male of forty-five years of age admitted with a seven days' history of acute obstruction whose etiology was obscure. Exploration revealed a true interstitial hernia with a loop of proximal ileum angulated within a midline pouch of peritoneum presenting beneath the rectus muscle. Both of these cases were admitted in shock from neglected obstruction of undetermined origin and both died within twenty-four hours after operation, thus corroborating established conviction that this type of hernia frequently escapes detection and, in consequence, is characterized by a relatively high mortality rate.

The third member of the group of rare obstructive hernias, representing an unusual type of internal hernia, is of particular interest academically as well as clinically.

CASE 1.*—The patient, a male aged thirty years, was admitted as an acute emergency under diagnosis of perforated duodenal ulcer. During the preceding five years he had complained of chronic indigestion accompanied by epigastric pain which usually appeared one hour after meals. Four hours before admission he developed an acute severe pain in the umbilical region accompanied by persistent vomiting. Bowels moved shortly before admission, and no blood was noted in either vomitus or stools.

On examination, the patient presented an anxious, grayish facies. The pulse rate was 68, temperature 98.6 degrees, and blood count showed 27,200 white blood cells with 96 per cent polynuclears. The abdomen was generally rigid and tender throughout and no masses were palpable. Rectal examination was negative, and there had been no bleeding per rectum.

Operation, carried out under diagnosis of perforated duodenal ulcer, disclosed a left paraduodenal hernia comprising the entire ileum, a portion of jejunum, cecum, and ascending colon. The hernial sac, as large as a human head, occupied the left gutter, and loops of black, hemorrhagic intestine could be seen through the transparent posterior peritoneal covering. On opening sac through incision over its summit, it was found that the hernia was complicated by torsion of an elongated, primitive mesentery resulting in gangrene of the distal ileum, cecum, and ascending colon (Fig. 1). This condition was treated by resection of the involved intestine, end-to-end anastomosis, replacement in retroperitoneal sac, and closure of incision through posterior peritoneum. Reduction of the hernia with obliteration of paraduodenal fossa was indicated as a concluding step, but because of patient's poor condition, this procedure was abandoned and operation concluded. Postoperative course was satisfactory, and patient was discharged at the end of the third week. This patient has been under close observation during the ensuing eleven years and since the persistence of the retroperitoneal hernia has given rise to no adverse symptoms, reoperation for the purpose of reduction of the hernia with obliteration of the paraduodenal fossa has been indefinitely postponed.

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INTERNAL HERNIA

The term internal hernia should properly include all those protrusions of intraabdominal contents through intraperitoneal pouches or openings of congenital or traumatic origin, thus contrasting with the common varieties of external hernias which take place through defects in the retaining wall of the abdomen. Although notably rare in

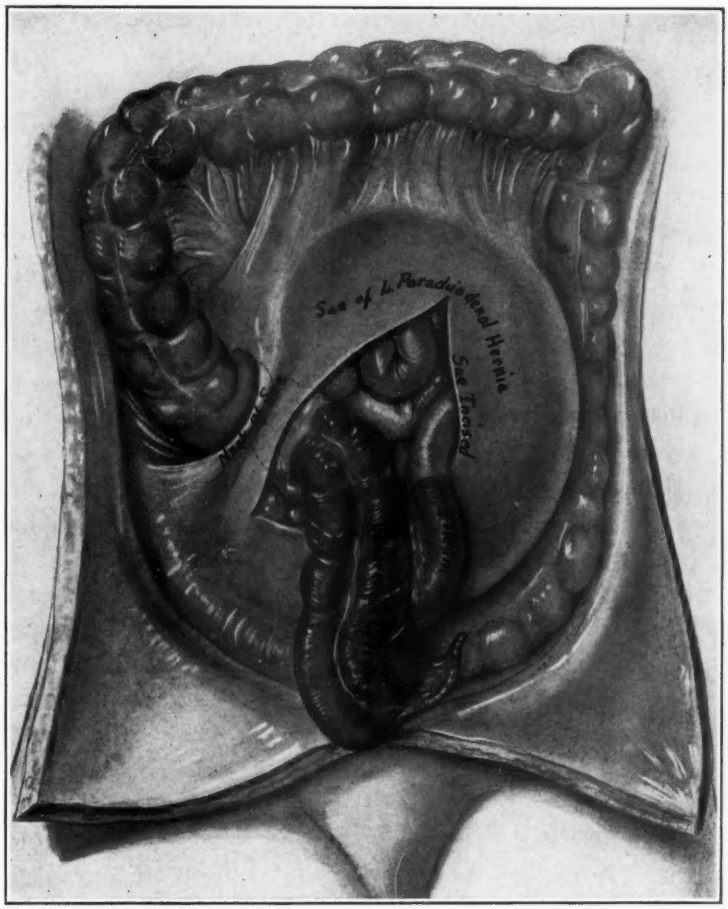

Fig. 1.—Drawing of left paraduodenal hernia (Case 1) to show neck of sac, exploratory incision of sac, and gangrenous segment of ileum and colon.

comparison with the external types, the internal hernias assume considerable clinical significance in consequence of the high incidence of acute intestinal obstruction which they exhibit. Short[1] mentions seven common types; viz., the retrocolic, the ileoappendicular, the intersigmoid and those through the foramen of Winslow, the transverse mesocolon and those into the paraduodenal fossae, right and left. Of this group, the last named, the paraduodenal, or retroperitoneal, hernia is of greatest importance from the clinical viewpoint and is differenti-

ated from the other types of internal hernias, not only by its clinical manifestations, but also by the fact that it represents a congenital anomaly.

Although Neubauer, of Leipzig, recorded in 1776 the first case of retroperitoneal hernia discovered in the course of a routine autopsy, and Landzert in 1871 for the first time described the anatomy of the paraduodenal fossae, this subject remained an academic one until 1906 when Moynihan established its status as a clinical entity and standardized the confused nomenclature and knowledge relating to this condition. He described nine different types of fossae about the duodenum in which hernia may take place and reported seventy-four examples of these hernias collected from the literature up to that time. Meanwhile a rapidly increasing number of case reports have swelled this figure so that approximately 200 cases are at this time available for study. We have recently had occasion, in another connection, to make a careful analysis of a group of these cases and were able to establish facts which have a bearing upon the present discussion of hernial obstruction. The series of paraduodenal hernias under consideration comprises 85 cases of which 59 per cent were males and 29 per cent females. The average age was 29.5 years, the youngest being aged 12 months, and the oldest 84 years. These cases were classified under two heads: (1) The silent or autopsy group which included all those instances in which the hernia was an accidental discovery during routine autopsy and in which there was no evidence that it had produced symptoms during life. In this group there were 59 cases, or 69.5 per cent of the series. (2) The acute clinical group comprising those cases treated or operated upon for symptoms incident to the presence of the hernia, or whose death, if untreated, was directly attributable to the hernia. In this group there were 25 cases, or 30.5 per cent of the series.

It is, therefore, apparent that a large percentage of the paraduodenal hernias are clinically silent, and since they do not present externally, the incidence of this condition cannot be accurately determined. It is furthermore evident that these hernias assume major clinical importance only where their benign course is complicated by some degree of incarceration or strangulation. In this latter respect, these hernias exhibit a close analogy with their external prototypes, but it is to be noted that, whereas, the incidence of these obstructive manifestations in the external hernias is less than 4 per cent, the corresponding figure for the paraduodenal hernias is 30.5 per cent.

Of the nine duodenal fossae described by Moynihan, one only—the left paraduodenal fossa of Landzert—will be considered at this time; first, because it is concerned in 70 per cent of all duodenal hernias, and

second, because Case 1 reported above is an example of this type of hernia. This fossa is formed just to the left of the terminal portion of the duodenum from the anterior surface of which two thin non-vascular folds pass laterally to merge with the posterior parietal peritoneum along the line of the inferior mesenteric vein. There are thus formed a superior and an inferior crescentic fold which coursing laterally become continuous with a falciform fold of posterior peritoneum raised by the inferior mesenteric behind. There is thus formed, to the left of the duodenojejunal flexure, a peritoneal pouch whose orifice looks to the right and whose anterior crescentic neck is

Fig. 2.—Drawing of dissection of left paraduodenal fossa. Inferior fold has been incised and reflected to show vascular relations at neck of fossa.

formed by the peritoneum-invested inferior mesenteric vein. (Fig. 2.) Herniations into this left paraduodenal fossa of Landzert insinuate themselves behind the inferior mesenteric vein and present as a true peritoneal sac in the retroperitoneal region of the left gutter.

Diagnosis in the obstructive type of left paraduodenal hernias presents obvious difficulties, and it is not remarkable that in the series cited there has been but one correct preoperative diagnosis recorded previous to 1906—that of Sherren, of London. Subsequently, in 1921, Kummer[2A] succeeded in making a preoperative diagnosis of left paraduodenal hernia by x-ray. The symptoms parallel closely those of the obstructed

external hernias except that they are not associated with or identified by the usual external hernial mass. A large proportion of cases give a history of recurrent attacks of indigestion associated with occasional attacks of epigastric pain and marked constipation, which may be intermittently replaced by periods of diarrhea, all of which express varying degrees of incarceration or partial obstruction. With the onset of complete obstruction, two very significant signs appear in more than 50 per cent of the cases, and when observed in conjunction with the above history, should establish the diagnosis. The first is due to the presence of the large retroperitoneal hernial mass which appears in the mesogastrium slightly to the left as a smooth, cystlike, globular tumor, varying in size from that of a grapefruit to an adult head. This mass is somewhat movable, yields a tympanitic note on percussion, and on auscultation over it, clear intestinal sounds can be heard. The second diagnostic sign results from a combination of mechanical and anatomic factors at the orifice of the paraduodenal fossa which becomes the constricted neck of the hernial sac. As the hernia increases in size, the thin crescentic margin of this orifice is exposed to the effects of stretching and dragging by the massive sac contents, so that the inferior mesenteric vein coursing along the sharp anterior margin of the constricted neck suffers a degree of compression which produces mechanical stasis and back pressure in the inferior mesenteric venous system, and this is expressed clinically by the acute development of large internal hemorrhoids or bleeding from the lower bowel. It seems justifiable to conclude, therefore, that a preoperative diagnosis of paraduodenal hernia should be made with a reasonable degree of certainty when the clinical evidences of acute intestinal obstruction are associated with the presence of a large, globular mass in the left mesogastrium and accompanied by lower bowel hemorrhage.

Space will not permit a consideration of the etiology of these hernias, but it may be stated that the theory of congenital origin of the paraduodenal fossae and associated phenomena is well supported. During the conversion of the embryonal status of the gastrointestinal tract, characterized by the primitive dorsal mesentery, into its permanent adult relation, the cecum migrates upward from its left lower abdominal position toward the left upper abdomen, then progresses across the epigastrium to eventually become fixed by fusion of fetal mesenteries in the right lower quadrant. It seems quite clear that congenital anomalies, such as the fossae about the duodenum and the persistence of fetal mesenteries, represent the failure of the peritoneum to properly adjust itself during this process. It is interesting to note the frequency with which the fetal type of mesentery with mobile cecum or colon and incomplete rotation of the large intestine are reported in conjunction with retroperitoneal hernias. This fact is of

importance clinically because it is this fetal type mesentery with the wandering cecum and colon which makes possible the herniation of a large proportion of the intestinal tract as noted in Case 1.

The sac contents in the paraduodenal hernias vary from those of a few inches of the jejunum to massive inclusions of the entire small intestine with a portion of the large, as described under Case 1. Obstruction may occur in all types, but the larger hernias provide the true predisposing factors; viz., (1) a large hernial sac with comparatively small, unyielding vascular margin favoring obstruction at the neck; (2) long fetal mesentery with mobile cecum and colon predisposing to volvulus (Case 1); (3) intrasaccular adhesions leading to simple obstruction.

Although the principles governing the treatment of the external hernias (viz., [1] reduction of the hernial contents and [2] obliteration of the sac), theoretically hold good for the paraduodenal hernias, it may be recalled that the latter are clinically silent until their presence is signalized by the development of such complications as incarceration, volvulus, or strangulation, and hence when the problem of therapy arises it involves the treatment of one of these complications. The constricted neck of these hernias is a frequent site of obstruction and constitutes a major problem because the presence of important vessels in the free edge of the neck contraindicate incision of the constriction at this point. Under these conditions, opening the sac itself will frequently permit a retrograde reduction of the contents, and this same maneuver is indicated in case of volvulus, incarceration, or strangulation within the sac, and particularly when resections of gangrenous intestine become necessary. Partial excision of the sac, obliteration of the cavity by suture, or closure of the orifice are advisable after reduction of the hernial contents, due consideration being given to the proximity of important neighboring vessels. These patients are usually received in some degree of shock and an operative mortality rate exceeding 50 per cent was recorded in some of the early series. In the meantime, the modern policy of prompt intervention in cases presenting symptoms of intestinal obstruction has gradually reduced this rate so that it now approximates the figure which obtains for cases of intestinal obstruction in general.

There were three instances of partial enterocele (Richter's hernia) recorded in the series of 130 cases of obstructive hernias. The first instance complicated a femoral hernia in a female of fifty-five years of age and involved a portion of the wall of the ileum in a gangrenous process requiring resection. The second case occurred in a female of forty-one years of age in conjunction with a ventral hernia. Reduction was accomplished without the necessity of resection. The third

instance was observed in a male of sixty years of age in whom a knuckle of ileum became involved in a recurrent inguinal hernia. The obstructed loop was freed through a right rectus incision and returned to the abdomen without resection. In all three cases the obstruction was complete and of less than three days' duration. There were no deaths in this group.

Chart I records by decades the age incidence in 130 cases of acute intestinal obstruction due to hernia and graphically emphasizes the exceptionally high incidence of this condition in the first ten years of life (21, or 16.1 per cent of the cases) with an abrupt fall in the second decade, followed by a gradual rise to a maximum (30, or 23 per cent

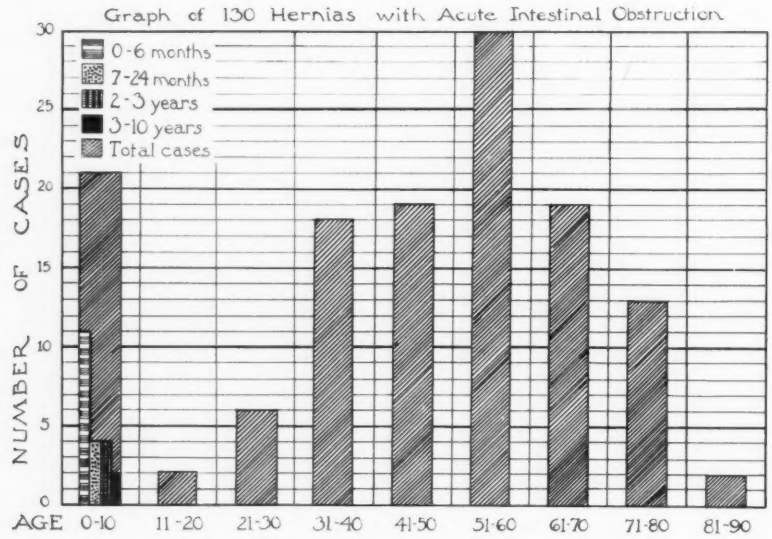

Chart I.—Age incidence by decades in 130 hernial obstructions with insert graph analyzing cases occurring in the first decade.

of the cases) in the sixth decade. The insert graph in the first decade is of particular significance and indicates that of the cases noted during this period, 11, or 52.3 per cent, occurred during the first six months of life; 15, or 71.4 per cent, took place before the second year; and 19, or 94.2 per cent, before the third year. The unusually high incidence of hernial obstruction noted in the first decade has usually been explained on a basis of certain anatomic and mechanical factors peculiar to children, but in our opinion this incidence merely expresses the conventional ratio of obstructive complications to be anticipated in a decade which boasts of the greatest hernial frequency. In a study of 10,000 hernias, Berger[2] showed graphically that 75 per cent of hernias made their appearance during the first decade, after which the curve of frequency fell sharply through the second and

third decades so that, if the incidence of obstruction in this group remains consistently proportional, its curve should roughly parallel the curve of frequency.

Chart II compares the age incidence in both sexes and brings out the facts that (1) the high incidence of obstruction in the first decade takes place in the male sex, (2) that hernial obstruction in the female sex is extremely rare before the third decade, and (3) that after the second decade the incidence curves of hernial obstruction in the two sexes run roughly parallel courses.

Complications, such as incarceration and strangulation, arising during the first decade present special features which distinguish them from the analogous adult condition. These complications are undoubt-

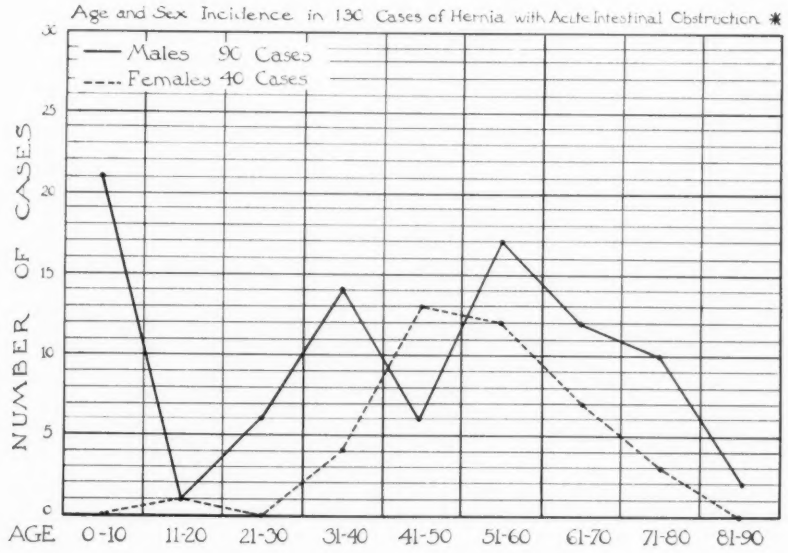

Chart II.—Comparison of the age incidence of hernial obstruction in both sexes.

edly most common in the first year and are probably related to attempts at natural closure. The small irreducible hernias are more dangerous than the larger types because they are most often associated with circulatory disturbances. Irreducibility is, however, particularly innocuous to children, and even with intestine in the sac, obstructive symptoms are infrequent. The rarity of true strangulation at this period of life is confirmed by the investigations of Estoei[3] who showed that among 139,000 hernias in children there was not a single herniotomy done for strangulation. Fraser[4] found in an analysis of 10,000 cases at the Edinburgh Children's Hospital that there was no instance of complete strangulation and gangrene such as is seen in adults. Out of the total series, there were fourteen cases of irreducible hernia in which there was sufficient interference with blood supply to justify the qualification of strangulation, and in these extreme in-

stances the vascular changes were limited to patches of necrosis. Analysis of our own series of obstructive hernias occurring in the first three years shows that all were males, all had inguinal types of hernia, thirteen were obstructed at the external ring and five at the internal, while the small gut was involved in thirteen instances and the large in four. There was no instance in which gangrenous intestine required surgical attention; there were no deaths; and the average duration of obstruction was less than twenty-four hours. This last figure, compared with the average duration of obstruction in the series of 130 cases of all types and ages, suggests that the rarity of true strangulation in children is due less to anatomic and mechanical factors and more to the spirit of promptness which brings these children to early operation.

In general, the symptoms induced by hernial obstruction were conventional and require little comment. An acute onset of pain associated in the external hernia, with an enlarged, tense, irreducible, and sometimes inflamed hernial mass, was quite constant. Nausea, vomiting, and constipation were usually observed, but distention and other abdominal evidences of obstruction were as a rule conspicuously absent due to the infrequency of low gut obstruction. Temperatures, with few exceptions, were normal or subnormal and white blood counts presented, in most instances, normal or very slightly elevated figures.

Of greatest significance in the clinical picture is the appearance of a condition called shock characterized by an ashy pallor, cold clammy skin, a rapid pulse of small volume, and hypotension. This condition was most commonly observed in elderly patients with prolonged obstruction, but it was not infrequently noted also in relatively young subjects whose hernias had come down for the first time and in whom the obstruction was of short duration. In three instances this type of shock induced a moribund state which precluded operation and led to a mortality in each case, while among the 38 deaths which occurred among the patients operated upon, 39 per cent were ascribed to this cause.

This same shock syndrome, which is recognized as an important feature in severe trauma generally, has attracted the interest of many able investigators and many divergent theories have been advanced to account for it. At the present time there is quite general agreement that diminution of the circulating blood volume is an essential part of the shock picture, but there is a wide difference of opinion as to the cause of this diminution. Crile and his associates maintain that prolonged sensory stimulation flowing from the traumatized regions produces exhaustion of the vasomotor center which leads first to a

fall in blood pressure and which is followed by an accumulation of blood in the large veins. Others hold to the theory that shock is associated with constriction of the arterioles which expresses plasma from the blood stream and in this manner brings about a decrease in the volume of circulating fluid. Cannon[5] believed that the persistent fall of blood pressure following trauma was due to the liberation by the traumatized tissue of a histamine-like depressant. Blalock and Bradburn,[6] working with dogs, observed that the oxygen content of blood from the femoral vein in traumatized extremities was consistently higher than the controls and suggested that a local accumulation of blood occurs in the traumatized area. Subsequently, Blalock[7] estimated the blood loss in traumatized areas and presented evidence which indicated that the low blood pressure noted after trauma to the extremity of an experimental animal is due to loss of blood into this traumatized area. Finally, this same observer,[8] noting that prolonged trauma to the intestinal tract resulted in a marked decline of blood pressure, studied in the same manner the effect of trauma upon the intestines and concluded that the loss of fluids from and into the traumatized intestinal segment was the chief cause for the reduction in pressure observed in these instances.

Among our cases of hernial obstruction, there were twenty-one instances in which varying degrees of shock were observed, and it is our conviction, based upon these observations, that clinically at least this condition differs in no important respect from the type of shock seen in severe trauma. As to the mechanism of its causation in hernial obstruction, it may be pointed out that incarceration or strangulation of an intestinal loop is associated with a degree of prolonged trauma; and it is tempting to assume, in the light of experimental evidence, that the consequent loss of fluid into and about the traumatized loop due to hemorrhage, capillary engorgement, and filtration of plasma is sufficient to produce the typical fall in blood pressure and shock.

TREATMENT

In reference to treatment, it is important to recognize that fundamentally hernial obstruction presents two problems for solution; the first, provided by the obstructed intestine, is local and primary; while the second, expressed in the form of constitutional effects of the obstruction, is general and secondary. To meet the first problem three possible indications are to be considered: (1) relief of obstruction; (2) treatment of the strangulated hernial contents; (3) repair of the hernia. Relief of obstruction is the primary purpose of operation; it is frequently a lifesaving measure and is occasionally the only step permissible where severe shock exists. Under these conditions opera-

tive procedure is limited to simple incision of the constriction combined with enterostomy in situ or with exteriorization plus enterostomy in those instances in which severe intestinal damage or ileus indicate the need of decompression and subsequent repair of intestine. Treatment of the hernial contents, the next indication, is second in importance only to the relief of obstruction and is reserved for those patients whose condition will tolerate more extensive procedures. At this point, decisions must often be made which tax the judgment of the most experienced operator—choice between reduction, exteriorization, and resection; determination of type of anastomosis; estimate of the patient as a surgical risk; etc. Each problem must be individually evaluated upon its own merits and only general, well-recognized principles can be laid down. Dark, congested, or even purplish gut whose serosa retains its normal gloss may usually be reduced without fear; whereas, the loss of this sheen with persistence of black discoloration after release usually indicates the necessity for resection. In the small gut, the aseptic type of end-to-end anastomosis is to be preferred; in large gut resection the side-to-side method is undoubtedly the safest; and in both a proximal catheter enterostomy should invariably be added for decompression. Prolonged stasis with ileus and dilatation may contraindicate primary anastomosis in some cases and some form of decompression-excision procedure on the Mikulicz principle may be required. In children, resection is accompanied by a high mortality and is to be employed only where indications are positive and definite. On the other hand, palliative enterostomy is poorly tolerated in early life and immediate resection is preferable to enterostomy.

Among our series of 130 cases of hernial obstruction, 13 cases had primary resections and of these 6, or 46.2 per cent, recovered, while 7, or 53.8 per cent, died. Of the latter, 6, or 85.5 per cent, were in femoral hernias whose contents in every instance was small gut. The anastomosis was of the end-to-end type in 4 of these cases and side-to-side in 2 cases. Exteriorization was employed in 7 cases with 6 deaths and 1 recovery.

In respect to the second fundamental problem—the constitutional effects induced by obstruction—we are convinced from a critical analysis of our deaths, that among our early cases this factor was improperly evaluated and that ill-advised haste has contributed materially to mortality rates. Subsequent experience has led to the paradoxic conclusion that the early obstructions are the logical operative emergencies, while the advanced cases admitted in shock merit delay provided that this period of delay is properly utilized. In other words, early operations on obstructions of short duration mean a minimum degree

of shock, a lower operative mortality, a limitation of progressive tissue destruction, and a decreased incidence of resection or enterostomy. On the other hand, hasty operation in advanced cases, complicated by typical shock, imposes a major surgical procedure upon a patient already overburdened by dehydration from fluid loss, distorted blood chemical values, gastrointestinal ileus, etc., and when disregarded preoperatively, these latter factors account for a considerable proportion of the surgical deaths.

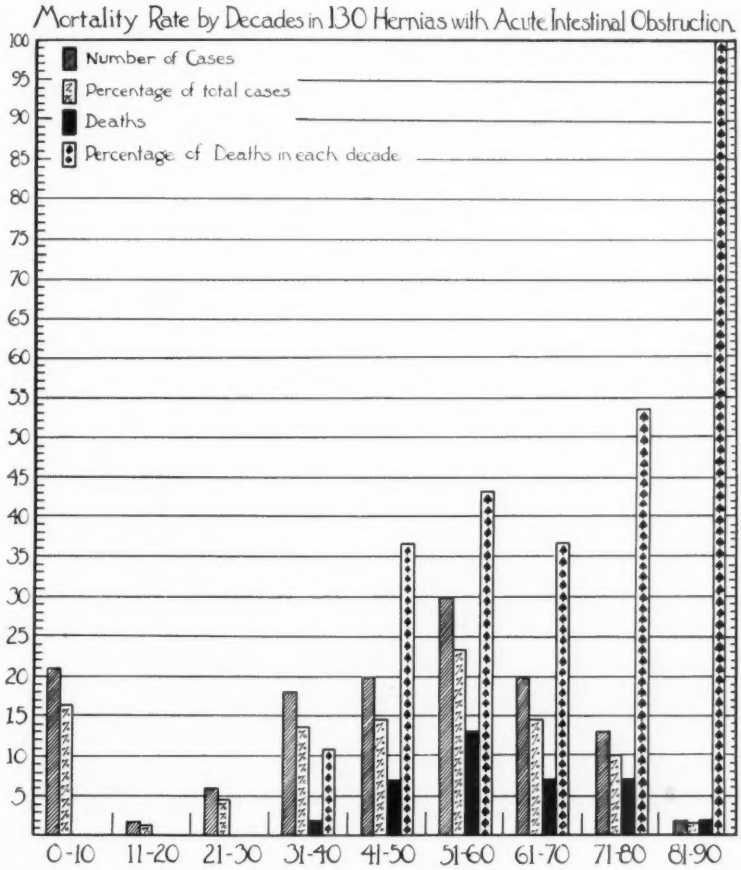

Chart III.—Graphic analysis by decades of the 38 deaths occurring among 130 hernial obstructions to show distribution and percentage distribution of deaths in relation to total number of cases.

Thus the ideal attack upon the constitutional phase of this problem suggests itself; viz., that regardless of the degree of local damage or the duration of obstruction, operation should await a preliminary period devoted to restitution, so far as possible, of perverted chemical and physiologic processes. Shock and dehydration being characterized fundamentally by diminution of circulating fluids, the first indication is the restoration of these fluids in a form which will remain longest in the vascular tree. Whole blood best meets this indication

and our preference at this time is for its administration by the citrate method. Gum acacia solution, when properly prepared, glucose and saline solution, or normal saline solution alone, are of considerable value in correcting distorted blood chemical values, raising blood pressure, and replacing fluid loss, but they are all of secondary choice because of their transitory effect. They are, therefore, best given in massive amounts by slow, intravenous drip. Decompression of the gastrointestinal tract is of equal importance as a preoperative measure in the presence of distention and vomiting, and the suction-siphonage method devised by Wangensteen[9] plays an extremely important rôle in this as well as in the postoperative period.

The treatment of the hernia in hernial obstruction requires little comment since it plays a small part in the problem after the obstruction has been relieved and the hernial contents cared for. In obstructions associated with gut damage, its repair must obviously be postponed, and even in those early cases where the damage has been minimal, ideal repair must be attempted with caution since it has been shown[10] that in strangulated hernias a grossly intact intestine may be permeable to bacteria which may frequently be cultured from the sac fluid.

As previously noted, there were 38 deaths in this series establishing the mortality rate of 29.2 per cent. Chart III analyzes this group by decades and shows that, beginning with the fourth decade, the actual number of deaths rose gradually to reach a maximum in the sixth decade, while the percentage death rate shows a gradual rise from the fourth to the ninth decade where it reaches its maximum. Of the 38 deaths, 35 were operative and 3 died without operation. Twenty of these cases were males and 18 were females. Femoral hernia led the list as a cause of death with 15 cases; inguinal hernia was second with 13 cases, and all other types combined numbered 10. The immediate cause of death was ascribed to shock in 15 cases (39 per cent) of this group; to pulmonary complications in 8 cases; to cardiocirculatory failure in 5 cases; to peritonitis in 2 cases; and to miscellaneous factors in 8 cases. The average age of the patients who died was 60.2 years.

In conclusion, we may briefly summarize the results of our study of 130 cases of hernial obstruction as follows:

1. Hernia, inclusive of all types, comprises 6.5 per cent of all general surgical hospital admissions; 4 per cent of hernias are at some time or other complicated by obstruction; and this 4 per cent is responsible for 54 per cent of intestinal obstruction from all causes.

2. Obstruction due to femoral hernia shows the highest figure for average age of onset, duration of symptoms, and mortality, while indirect inguinal shows the lowest.

3. Although hernial obstruction shows a high incidence in the first decade, and 94.2 per cent occur before the third year, there was no single instance in which resection of gut was required.

4. Early operation and release of obstruction constitute the primary objectives of surgical treatment.

5. Shock is the most serious complication of hernial obstruction, 39 per cent of deaths being ascribed to this cause alone.

6. The mortality rate in 13 primary resections was 53.8 per cent while 7 exteriorizations showed a mortality rate of 85.7 per cent.

7. The average age for the series was 44.1 years, while the average age in 38 cases which died was 60.2 years.

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INCIPIENT VOLVULUS OF THE CECUM ASSOCIATED WITH LEFT-SIDED COLON

Report of Case, with New X-ray Sign Obtained by Barium Enema

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AUTHORITIES agree that a torsion of the intestine on its mesenteric axis must be of at least 180 degrees to constitute a volvulus, since pathologic changes rarely begin before that stage is reached. However, in the course of an exploratory laparotomy, one sometimes encounters a condition that may be regarded as an initial stage of volvulus; i.e., a twist of less than 180 degrees. Such a discovery is comparatively rare, and as very little can be found in the literature regarding this condition, the following case may be of interest as a typical illustration:

Case Report.—T. C., white, male, twenty-six years old, electrician's helper. Admitted to Knickerbocker Hospital August 8, 1936, complaining of pains in the lower abdomen, of three hours' duration, accompanied by vomiting.

The patient had taken a bottle of citrate of magnesia several hours before onset to relieve constipation, and this had produced two good bowel movements. The pains, which were of a very severe, colicky nature, were more pronounced on the right side, but did not radiate. Sitting up or coughing increased the pain, but other movements had no effect. Patient had vomited food and greenish material twice in the past hour without relief.

Three months ago the patient suffered a similar attack, but without vomiting. The pain lasted for some three hours and then cleared up spontaneously.

Physical examination was essentially negative, except for the abdomen. Breathing was costal in type. There was a generalized moderate rigidity, most pronounced in the right lower quadrant. Tenderness on deep palpation over both lower quadrants, reaching its maximum over McBurney's point, and slight rebound tenderness in the right lower quadrant. There were no masses, and no distention was apparent. Resistance in both flanks, but no tenderness. Rectal examination negative. Soon after admission the patient vomited again, the vomitus consisting of an intestinal content typical of the early stages of obstruction.

Blood count showed 11,800 white blood cells; 78 per cent polymorphonuclears; hemoglobin, 98 per cent. Urine negative. Temperature and pulse were only slightly above normal.

The impression was one of acute appendicitis, or possibly a perforated peptic ulcer. Two and a half hours after admission an exploratory laparotomy was done under ether anesthesia.

Operation.—McBurney incision. On opening the peritoneum, a moderate amount of free, clear serous fluid was found. No ascending colon or cecum was seen in the normal position. Several loops of small intestine were encountered, which were for the most part collapsed. The incision was then prolonged downward in order

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to locate the appendix and the ileocecal junction. The appendix was finally found, pointing downward into the pelvis and covered by a greatly distended, plum-colored intestine, which proved to be the cecum. The appendix was gradually brought upward and seen to be moderately injected. At the same time it was noted that the cecum itself rotated, or turned, to a more normal position. The distention disappeared, and the small intestine that had been previously collapsed became moderately distended. Simultaneously the discoloration of all the intestines appeared to clear up with a restoration of the circulation. The torsion was apparently in a counterclockwise direction. After release, it was noted that the cecum and terminal ileum were attached by a long common mesentery, which allowed great latitude of movement.

After removal of the appendix in the usual manner and inversion of the stump, it was possible to demonstrate that the ileocecal junction was in an unusual posi-

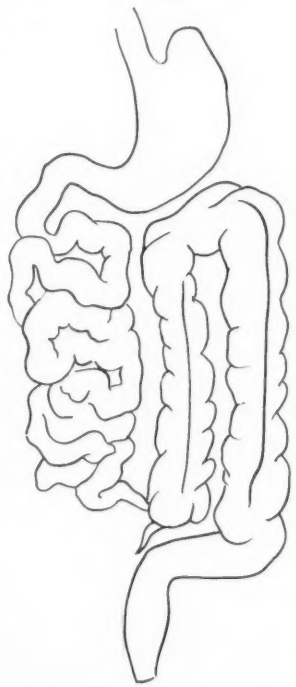

Fig. 1.—Schematic drawing indicating unusual ileocecal relations and sinistroposition of the colon as demonstrated both at operation and subsequent x-ray examinations.

tion. The small intestine extended from above at the right side of the abdomen to unite with the cecum below and to the left. Further investigation showed that the entire large intestine was also on the left side of the abdomen, as in the accompanying diagram (Fig. 1). As no further procedure seemed advisable at the time, the abdomen was closed without drainage.

The patient made an uneventful recovery, at no time during his stay in the hospital complaining of the pain which had caused him to seek admission. He was discharged on the twelfth postoperative day.

Before discharge, a barium enema was given by I. Schwartz, with the following findings: August 19, 1936: All of the colon visualized was found in the left of the abdomen. At the splenic flexure, several redundant loops of colon were visualized, lying below and to the left of the flexure. One loop of gut was traced downward

to the midline from the region of the splenic flexure. Its terminal portion was concealed by overlying loops of sigmoid. After expulsion of part of this barium, the x-ray appearance did not change appreciably. Twenty-four hours later, no spontaneous bowel movement having occurred, a soapsuds enema was administered and all the barium expelled from the colon.

Ten days later, following the patient's discharge from the hospital, a gastrointestinal series confirmed the above findings. Neither fluoroscopic examination nor roentgenogram revealed any abnormality of the duodenum, and as this organ was not explored at the time of operation, it was not known whether it had a distinct mesentery such as might be expected with nonrotation of the colon.

The findings in this case of a left-sided colon and a volvulus of the cecum in an initial stage of less than 180 degrees were both so unusual as to suggest a search of the literature to see if any similar cases had been recorded. The nearest parallel discovered is that reported by Fécan,[1] as follows:

A woman of twenty-one years of age had been troubled with intermittent digestive disturbances for two or three years. She had nausea, vomiting, and habitual constipation but had never suffered an acute attack of appendicitis. Repeated physical examinations failed to reveal any masses. To verify a tentative diagnosis of chronic appendicitis, a radiographic examination was made, which disclosed a transposition of the large intestine to the left of the median line and at the same time an abnormal development of one loop of the sigmoid. It could not be determined, however, precisely where the internal border of the cecum was, or if it was diseased. Only one thing seemed clear—that the point of tenderness of the right iliac fossa was not due to the appendix.

A barium enema two days later showed exactly the same abnormal topography of the large intestine. One could see filling up immediately an enormous sigmoid loop, which extended up into the abdomen to the level of the second lumbar vertebra. Instead of being stretched out at length, as is usual in similar cases, it was turned at an acute angle to the left of the median line. Next, at the left of this double gunbarrel, the descending colon filled in its turn, became dilated, and the splenic flexure was visible in its usual position. At last, a fourth segment filled up, at the right of the sigmoid loop this time, very oblique below and at the right, extending considerably beyond the median line because of the distention. It showed the transverse and ascending colons, their angle being almost obliterated. The cecum was superimposed on the rectum and on account of the sigmoid loop could not be seen very distinctly. After the injection of at least 1 and ½ liters of opaque liquid, four great shadows could be seen juxtaposed from right to left, leaving the right iliac fossa transparent. Thus two angles were delineated, one in the other, that of the sigmoid loop on the inside and that of the colon on the outside.

The examination of this patient relieved her of the incomplete torsion of the intestine.

Since, in our case, the patient had no recurrence of symptoms for seven weeks and was apparently cured, it was believed that the simple untwisting of the partial torsion had brought the same happy results as in Fécan's case. However, on October 7 a sudden flare-up occurred, and the patient was readmitted to the hospital.

Second Admission.—Since his discharge on August 20, the patient had been troubled with chronic constipation. Bowels had been moved regularly with mineral oil. The day before admission, October 6, he noticed dull pains in the lower abdomen, which gradually increased. Vomiting began two or three hours later and had continued intermittently. The morning of admission he suffered a violent attack of colicky pain, which he said was the same as he experienced prior to his first admission. The pain did not radiate. He had a good bowel movement the day before but none on the day of admission.

Abdominal examination was essentially negative, except for an indefinite mass in the suprapubic region. No visible peristalsis; no localized tenderness or rigidity; no distention. Patient catheterized after voiding, but no urine obtained. High colonic irrigation resulted in the passage of a few fecal particles and some flatus and diminished the circumference of the abdomen 1 cm. Temperature, 98; pulse, 72; respiration, 18. Blood count, 10,000 white blood cells; 88 per cent polymorphonuclears.

After careful consideration, it was decided that no immediate surgical intervention was indicated until further x-ray study by means of barium enema could be made. This was done on October 8, with meager results. The column of barium was not seen to reach beyond the portion of the large intestine corresponding to the transverse colon. Considerable gas appeared distributed in the right lower quadrant and suprapubic region of the abdomen in a hollow viscus which suggested by its conformation part of the large intestine (ascending colon and cecum) (Fig. 2). No great distress was evident following the barium enema, and the patient slept comfortably all night. He was examined the next morning by several members of the staff, and the roentgenograms were considered. It was decided that there was still no indication for an emergency operation. Vomiting continued intermittently. Clyses were given and Wangensteen suction siphonage afforded relief.

On the morning of October 9, a soapsuds enema produced a considerable return of barium. No change was noted in the abdomen at any time during the day. In discussion of the case at a staff conference, perforation seemed an unlikely contingency, and plans were made for an operation of choice, preferably a cecopexy.

A 5:30 A.M. on October 10, however, the patient was awakened by a sharp generalized pain followed by vomiting. The pain was particularly marked about the navel. The abdomen became boardlike throughout and tympanic. Liver dullness was partially obliterated. Temperature, 103.4° F.; respiration, 36; pulse, 132; blood count, 7,500 white blood cells; 93 per cent polymorphonuclears. Impression: rupture of a viscus, probably due to volvulus with gangrene.

Operation was begun at 8:00 A.M. *Findings:* The peritoneum was darkened, and when opened a large amount of foul-smelling gas escaped. The peritoneal cavity was loaded with liquid fecal matter and barium. The caput coli presenting in the wound was dilated. This collapsed and a clockwise torsion of the segment of more than 180 degrees became apparent. In reducing this torsion, a gangrenous area 7 or 8 cm. in diameter on the right lateral and anterior aspect of the cecum, about 4 cm. distal to the ileocecal junction, was uncovered. At its center there was a perforation approximately 1½ cm. in diameter.

Procedure.—Lower midline incision of 15 cm. Fecal matter and barium suctioned out and removed with wet sponges. Perforation temporarily closed with curved hemostat. Gangrenous area of cecum exteriorized, and healthy cecal edges sutured to the edges of the peritoneum. Penrose drain inserted into pelvis and wound left open. Continuous intravenous infusion of glucose and saline started.

The operation took twenty minutes, but as the patient was already in shock, with a weak, thready pulse, he did not rally. During the day the infusion was con-

tinued, forty drops per minute, and coramine and digalen injections were given, together with an adequate amount of morphine. Despite all these measures, the temperature continued to rise to 107.6°; and at 2:00 A.M. on October 11, the patient expired.

A partial postmortem examination of the wound was made by Adams, with the following findings:

A.

Fig. 2.—*A*, Barium enema five minutes after injection, showing the splenic flexure as a fixed point at the top and the blocking of the column of barium somewhat lower, to the right; also large gas bubble showing site of the volvulus. *B*, Barium enema four hours after administration, showing gas bubble even larger than in *A*, and typical conformation of the large intestine.

Postmortem.—There was about 500 c.c. of dark fluid having a fecal odor in the peritoneal cavity. There were numerous fresh adhesions about the intestines. The cecum was dilated to twice its normal size, and there was a circumscribed area of gangrene on its anterior surface about 8 cm. in diameter, in the center of which was a perforation about the size of a nickel. A markedly elongated common

mesentery was attached to the cecum and terminal ileum, allowing a wide range of movement to both structures. The terminal ileum entered the cecum from the right. About 1½ feet from the ileocecal junction, there was an old adhesion of the terminal ileum to the parietal peritoneum on the lateral abdominal wall covering the right iliac fossa. The ileum was twisted upon itself at this point.

Fig. 2B.—(*See opposite page for legend.*)

The stomach and duodenum were apparently normal, although the latter appeared to be somewhat more freely movable than usual. Whether a mesentery was attached to the duodenum could not be made out. There was no structure suggestive of a greater omentum.

The ascending colon was located approximately in the midline, running upward to about the level of the inferior border of the stomach, where it was fixed firmly to the posterior abdominal wall. The intestine was somewhat constricted at this point. It then ran diagonally to the left upper quadrant to form the splenic flexure. The descending colon and sigmoid were normal in appearance, course, and relations.

This is a case in which a few comments after the unfortunate event may serve as forewarnings to other surgeons confronted with a similar situation:

The recurrence of the same typical colicky pain as on the first admission should have constituted a definite warning that volvulus was present. This probability was thoroughly considered by the writers and other members of the surgical staff, but since the patient's symptoms were more or less chronic, it seemed advisable at the time to form a definite plan for the relief of his condition based on the results of another barium enema. In the meantime, the temperature, pulse, respiration, and blood count were so nearly normal as to lull one into a sense of security regarding any impending catastrophe, such as gangrene or perforation.

The barium enema itself evidently did not have the beneficial effect noted in the case reported by Fécan, but on the other hand it may not have greatly aggravated the situation. The findings at operation and autopsy would seem to indicate that an impaired circulation of the cecum supervened at some time between the patient's admission to the hospital on October 7 and the onset of his acute symptoms at 5:30 A.M., October 10. Whether this was entirely due to the incipient volvulus or partly to the anomalous blood supply of the cecum, whereby one of the terminal blood vessels became twisted and occluded as a result of torsion of the intestine and adhesion of the terminal ileum, is impossible to determine. It seems perfectly clear that only one such blood vessel was affected, as the area of gangrene was definitely demarcated and the perforation occurred at its center.

In view of the operative findings, it is necessary to review the meager results of the barium enema administered on the second admission. Quite evidently there was an obstruction to the flow of barium into the cecum, due to the torsion of the intestine, and the x-ray revealed a distended cecum with a large gas bubble in the hypogastrium and somewhat to the right of the portion of the intestine at which the column of barium was blocked in its progress. The significance of this finding was not entirely lost upon the writers, and now, in the light of the subsequent catastrophe, it seems to have been the final indication that operation should not have been delayed.

CONCLUSIONS

The finding of a volvulus of the cecum in its initial stages, with a clearing up of symptoms on simple untwisting, appears to be sufficiently unusual to prompt a report of the case. A rather extensive review of the literature had brought to light only two similar instances: the one recorded by Fécan[1] and another by Negroni.[2]

In our case, no attempt was made at the time of operation to correct the malposition of this freely movable segment of intestine because of the acute symptoms then prevailing. There followed an interval of about seven weeks during which the patient was free from symptoms, other than chronic constipation. Then a flare-up occurred, accompanied by an ill-defined mass in the suprapubic region. While investigation was being made to determine a more definitive method of surgical interference, conservative treatment resulted in gangrene and perforation, from which the patient died.

Sign.—The authors believe that a new diagnostic sign of great importance has been established for volvulus of the cecum: When in the course of a barium enema, the column of barium is unable to pass beyond a certain point in the region distal to the cecum; i.e., in the ascending or transverse colon (organic lesions being excluded) so that the cecum itself cannot be properly delineated in its haustral markings by the barium, and when at the same time a large collection of gas appears at the site of the cecum indicating its patency and conformation, either in the midline or slightly to the right of it, as in Fig. 2, this is a clear sign that volvulus of the cecum has taken place and is a definite indication for emergency operation.

Holubec[3] reported confirmation of a diagnosis of cecal volvulus by means of a lateral x-ray view, but in our case it is questionable whether the lateral view, taken before the barium enema, would have given similar results.

In view of this experience, the authors believe that any known case of nonrotation of the large intestine with similar indications should have immediate surgical intervention, with an operative procedure designed to fix the loose segment involved.

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PANCREATIC JUICE AS A FACTOR IN THE ETIOLOGY OF GALLBLADDER DISEASE

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IN STUDYING the literature on the subject of the etiology of gallbladder disease, only a few references are to be found, chiefly in the German, which deal with pancreatic juice as a possible factor. In contrast, it is the consensus of opinion that bile plays a distinct rôle in the causation of acute pancreatic necrosis. As one analyzes the latter postulation (i.e., that bile can be forced into the pancreatic duct and due to its action upon the pancreatic juice, or the pancreas itself, produce varying types of pancreatic disease), it would seem equally possible, or even probable, that pancreatic juice may enter the biliary ducts, and since the action of pancreatic juice may, under condition of activation, be very corrosive, lead to destructive changes in the biliary system. Therefore it may be pertinent to analyze further the problem with a view to clearing the hypothesis that pancreatic juice may be a factor to be considered in the etiology of gallbladder disease.

EMBRYOLOGIC CONSIDERATIONS

The liver with its ducts arises, in embryo, from a budlike projection of the main gastrointestinal stem. This diverticulum appears very early as a small budlike knob. The hepatic duct, including the larger intrahepatic ducts which secondarily grow and subdivide from it, and the ductus choledochus are the stem portions of the original hepatic diverticulum; the gallbladder and its cystic duct represent a special offshoot of the early diverticulum. The pancreas arises from two buds, the dorsal and the ventral. The smaller, or ventral, bud appears in the caudal angle between the gut and the liver diverticulum. Often this ventral pancreatic bud comes off directly from the hepatic tube, and as the common duct lengthens, it carries away with it the duct of that portion of the pancreas. (Fig. 1.) The dorsal and larger bud arises on the dorsal side of the gastrointestinal stem at a slightly higher level. With rotation of the gastrointestinal stem the smaller, or ventral, portion of the pancreas will be found lying adjacent to and inferior to the larger dorsal portion. Fusion of the two parts takes place while the duct of the dorsal pancreas is still the major duct. After fusion of the two portions of the pancreas, the ducts become connected, the ventral duct becoming the larger, the duct of Wirsung, while the dorsal atrophies to form the duct of Santorini. (Fig. 2.) This embryologic development forms the

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basis of reasoning relatively to adult anatomic arrangement of the ductus choledochus and the major pancreatic duct.

ANATOMIC CONSIDERATIONS

A study of the embryology clearly indicates that there must appear in a considerable number of cases a high union between the common and pancreatic ducts, since in those cases in which the ventral pancreatic bud comes off the hepatic diverticulum, it is carried away from the gastrointestinal stem as the common duct lengthens. Many anatomic

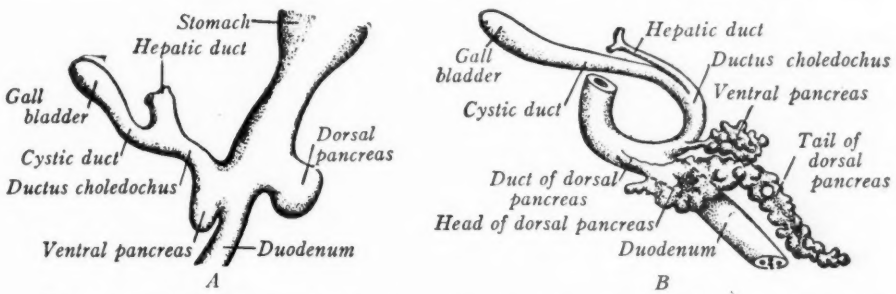

Fig. 1.—Reconstruction of the pancreatic primordia and main hepatic ducts in human embryo. (From L. B. Arey, Developmental Anatomy, Philadelphia, 1934, W. B. Saunders Company.)

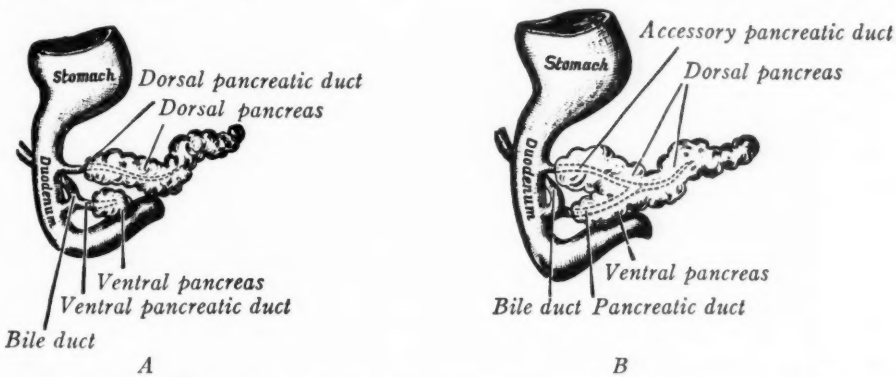

Fig. 2.—Origin and fusion of the two pancreatic primordia. (From L. B. Arey, Developmental Anatomy, Philadelphia, 1934, W. B. Saunders Company.)

studies have been made in an effort to determine the variations in the adult of the relationship between the common and pancreatic ducts at their junction near their exit into the duodenum, especially as to whether a common passageway could be demonstrated. All investigators have pointed out that, in the event of a common ampulla which is obstructed by a calculus, a continuous pathway between the pancreatic and biliary passages can exist only if the common ampulla has a length greater than the diameter of the duodenal orifice. In some carefully conducted studies on 100 necropsy specimens, Cameron and Noble demonstrated that in 65 per cent it was anatomically possible for a biliary

calculus lodged in the ampulla of Vater to cause a reflux of bile up the duct of Wirsung, or to cause a continuous pathway between the biliary and pancreatic systems. Westphal and Ruge stated that the percentages were 84 per cent and 75 per cent respectively, whereas Mann and Giordano found that in only 3.5 per cent is it anatomically possible for an obstruction at the duodenal end of the ampulla to convert the ducts into a continuous pathway. From these anatomic studies it must be concluded that in a large percentage of cases, in the event of an obstruction at the duodenal orifice by a calculus, a freely communicating passageway is formed between the biliary and pancreatic systems in which bile may enter the pancreatic ducts and pancreatic juice into the biliary system.

Other factors than calculi may play a rôle in the process of partial or intermittent duct occlusion at the papilla, thus forming a continuous pathway. Archibald believed that a spasm of the sphincter of Oddi might lead to increased pressure within the ducts, thus forcing bile into the pancreatic duct, causing pancreatic necrosis. This is controverted by Moynihan, and Mann and Giordano. Del Valle called attention to pathologic changes at the outlet of the ampulla, the result of stone, with infection, which he classified as: 1, hypertrophic papillitis; 2, atrophic papillitis; 3, erosive papillitis; 4, cholesterinic papillitis; 5, vegetative papillitis; and 6, phagedenic papillitis. Del Valle believed that these lesions accounted for the painful and distressing syndromes which occurred, at times, after gallstone operations and that the mechanism involved was spastic irritability of the sphincter with, in some instances, cicatrization and obstruction at the duodenal orifice.

If the hypothesis of a common pathway between the biliary and pancreatic systems is accepted, it becomes obvious that an interchange of pancreatic juice and bile in the ducts is possible. Nordmann ligated the ampulla and subsequently found bile in the dilated pancreatic ducts of the experimental animal. Delmore reported an instance in which, during an appendectomy, the gallbladder was squeezed in order to determine whether it would empty. Within twenty-four hours an acute pancreatitis developed. These observations would lend credence to the theory that it is possible for bile to enter the pancreatic system. Harms and Dragstadt have shown that during the height of digestion the pancreatic secretory pressure is greater than the biliary, therefore a reflux of pancreatic juice into the biliary ducts might be anticipated. We have shown that India ink introduced into the terminal end of the common duct of the dog will, at times, enter the gallbladder; hence, if pancreatic juice finds its way into the common duct, it, too, may ascend and enter the gallbladder.

If the assumption is correct that there may be an interchange of bile and pancreatic juice in the common duct and that pancreatic juice

may enter the gallbladder, it then remains to be proved what effect the pancreatic juice will have upon the ducts, liver, or gallbladder.

The entity, "acute nonperforative biliary peritonitis," was first described in 1910 by Clairmont and Haberer, their theory being that bile filtered through the wall of the gallbladder in the presence of duct occlusion. In 1918, Blad proved, however, by animal experiments, that the instillation of pancreatic juice into the gallbladder produced a biliary peritonitis in which the macroscopic appearance of the gallbladder was normal while microscopically extensive necrosis existed. We have corroborated this finding. The admixture of the bile with pancreatic juice leads to a "digestion necrosis" of the walls of the gallbladder, the bile passing through the wall of the gallbladder into the free peritoneal cavity. In 1923, Seifert observed a clinical case in which diastase was found in the bile, and in 1924, Schönbauer reported a case of gangrenous cholecystitis with a biliary exudate in the peritoneal cavity, trypsin being present in the bile. Bundschuh, in 1927, reported a case with a stone impacted in the ampulla and with a nonperforative biliary peritonitis in which pancreatic enzymes were isolated from the peritoneal fluid. In 1928, Ruppaner also reported a case of a stone impacted in the ampulla with biliary peritonitis in which pancreatic enzymes were found in the bile. In 1930, Popper reported a very interesting case:

Case Report.—A male, aged fifty-one years, was operated upon because of acute abdominal symptoms referable to the right side of the abdomen, a tentative diagnosis of acute appendicitis or acute cholecystitis having been made. Upon opening the abdomen, a large amount of a bile-stained fluid was found with fat necrosis of the omentum in the vicinity of the gallbladder. The gallbladder was found to be distended, large, and very tense, and the wall appeared to be edematous. The common duct was enlarged to the size of a thumb. There was no evidence of a perforation of the gallbladder; the stomach and duodenum appeared normal; and there was no evidence of tumor growth or obstruction. The pancreas was somewhat enlarged and firmer than normal, but no striking gross changes were noted. The gallbladder bile was turbid and dark green; no stones were found in the gallbladder or common duct, a large sound passing readily through the papilla. A cholecystectomy and drainage of the common duct were performed, and the patient recovered. The peritoneal exudate contained diastase values of $\mathrm{df}\frac{38}{30} = 1024$, and the gallbladder bile, $\mathrm{df}\frac{38}{30} = 4096$; trypsin could not be detected in the peritoneal exudate, which contained albumin; however, the gallbladder bile contained 9 units by the Fuld and Gross technic. At the time of operation, the blood diastase values were $\mathrm{df}\frac{38}{30} = 32$. The peritoneal exudate and the gallbladder contents were sterile, and the gallbladder, aside from being red, revealed neither gross nor microscopic changes. Numerous examinations were made of the biliary drainage in the days following the operation, but no evidence of pancreatic enzymes could be found; however, on the twelfth postoperative day, the drainage material was darker in color and cloudy.

Examination revealed diastase values of df $\frac{38}{30} = 4096$ and trypsin 8 units. These findings lasted only a few hours, and pancreatic enzymes were never again found in the biliary drainage.

Popper reasons that a small calculus became impacted in the papilla allowing pancreatic juice to find its way into the gallbladder, thus causing the pathologic picture; the calculus was forced through the papilla before the operation was performed, but an undiscovered stone remained in the dilated duct which later obstructed the papilla for a short period of time and was then expelled into the duodenum. Popper explains the process of acute nonperforative biliary peritonitis on the basis of obstruction of the biliary passage at the ampulla by stone; dilatation of the ducts follows, and there is a mixture of bile and pancreatic juices in the dilated ducts. Due to the excess of pressure in the pancreatic system, bile mixed with pancreatic juice enters the gallbladder and is rendered diffusible so that it passes through the wall of the dilated gallbladder. Bernhard, Dziembowski, and Brackertz have reported cases of nonperforative biliary peritonitis, and in 1934, Melchoir reported three cases. Bernhard stated, in 1931, that seventy cases had been observed and reported since the condition was first mentioned by Clairmont and Haberer. Birgfeld, in 1931, reported a typical case which followed a gastric resection for ulcer.

It must be conceded that whatever may be the mechanism of production of acute nonperforative biliary peritonitis, pancreatic enzymes have been found in the gallbladder contents and in the peritoneal fluid in these cases; also, that in some instances, notably those of Blad, Bundschuh, and Ruppaner, extensive necrosis of the gallbladder wall was present. Popper has, in my opinion, proved that the diastase is not carried to the gallbladder or peritoneum by way of the blood stream, but that it enters the gallbladder by a direct route, via the biliary and pancreatic passages. Since pancreatic enzymes are found in the gallbladder contents and peritoneal fluid, in cases of acute nonperforative biliary peritonitis, the question remains whether it may be the inciting factor in the production of changes in the wall of the gallbladder noted in some of these cases, and whether it may also produce other and less extensive pathologic changes noted in cases of acute, subacute, and chronic cholecystitis.

In 1931, I reported a series of experiments in which it was demonstrated that when pancreatic juice was introduced into the gallbladder, degenerative changes occurred in the wall of the gallbladder.

These experiments consisted of: (1) a single injection of pancreatic juice into the gallbladder through a hypodermic needle; (2) multiple injections of pancreatic juice into the gallbladder through a cholecystostomy tube; (3) a single injection of pancreatic juice into the cystic duct through a hypodermic needle; (4) a single injection of pancreatic

juice into the common duct through a hypodermic needle; and (5) the continuous introduction of pancreatic juice into the common duct by means of a special preparation (choledochopancreatic intubation).

In every instance the gallbaldder wall showed evidence of pathology; a normal gallbladder was never found in which the procedure or preparation used was considered successful. In every instance in which cultures were made, they were positive; the predominating organisms found were staphylococci and the colon group. The pathologic changes were characterized as degenerative and regenerative. In the accepted experiments, seventeen animals showed complete necrosis of the gallbladder, while eight showed incomplete necrosis with some evidence of reparative effort. In nineteen animals, the changes were classed as regenerative; these included hypertrophic, hyperplastic, and inflammatory reactions. The details of these experiments were published in 1931. It was noted "that the changes in the wall of the gallbladder have a degree of similarity, whether the pancreatic juice was introduced directly into the gallbladder through a needle, injected into the cystic duct or common duct with a needle, or continuously introduced into the common duct by means of a special preparation, choledochopancreatic intubation. It would seem that the sudden introduction of a relatively large amount of pancreatic juice, 20 c.c., is more likely to produce extensive changes than when smaller amounts are injected or when a choledochopancreatic intubation is used."

The conclusions drawn at that time were as follows: "A study of the microscopic changes in the various gallbladders would lead one to believe that under some conditions, the pancreatic juice brought about a prompt destructive action on the wall of the gallbladder as represented by a noninflammatory necrosis with edema. At other times, only partial necrosis took place with ulceration of the mucosa with, at times, rather marked changes in the muscular wall and thickening of the serosa. The changes in the muscular and serous coats appeared to be a reactionary response of the tissues to the destructive agent; inflammatory cells were called forth; edema was present; and in every way the process looked like typical inflammatory reaction."

In some instances, the process was typically a reactionary or inflammatory response as evidence that necrosis was either absent or present in a minor degree. There were all degrees of reaction from acute inflammatory manifestations with leucocytic infiltration and edema to the typical chronic changes characterized by lymphoid and papillary hyperplasia and cyst formation. The question arose as to whether the necrosis may not have been a postmortem process, either an autolysis or a postmortem digestion of the gallbladder wall by the pancreatic juice which had been introduced during the time of the experiment, and that the pancreatic juice did not attack the living tissues. We are convinced that in those

animals in which the autopsy was performed immediately after death and in those which were killed and the autopsy immediately performed, the necrosis was antemortem and associated with the introduction of pancreatic juice.

These experimental observations bear a close relationship with those of Blad, Schönbauer, Westphal, and Brackertz. Westphal used human duodenal juice which had been assayed as to trypsin and amylase content and passed through a Berkefeld filter to insure sterility. He introduced the duodenal juice into the gallbladder by a transduodenal cannulization of the common duct using a small catheter, thus obviating the possibility of trauma to either the ducts or gallbladder. He noted in two dogs, four or five minutes after the introduction of the duodenal juice, that the gallbladder showed evidence of gross changes. In one dog which died ten hours after the introduction of 8 c.c. of duodenal juice, the gallbladder was completely necrotic.

Andrews, Goff, and Hrdina have shown that when pancreatic juice was introduced into the gallbladder of the dog, pathologic changes occurred in every instance; however, when the pancreatic juice was passed through a Berkefeld filter, the changes were very much less. Brackertz concluded from his experiments that pancreatic extract mixed with colon bacilli when introduced into the common duct in the presence of stasis leads to necrosis of the wall of the gallbladder within twelve hours, but that pancreatic juice alone with stasis or pancreatic juice mixed with colon bacilli without stasis causes no changes in the wall of the gallbladder. These conclusions suggest that a bacterial contamination may be a deciding factor in the mechanism. Again we may turn our attention to work done on the relationship of the bile as a factor in the production of acute pancreatic necrosis. The observations of Nordmann have already been alluded to. With simple ligation at the ampulla, the pancreatic ducts were dilated; the contents consisted of bile-stained fluid, but there existed no necrosis of the pancreas. With the introduction of cultures of dog's feces into the gallbladder, prompt necrosis of the pancreas occurred. Binet and Brocq introduced sterile pus from an old empyema into the duct of Wirsung of an experimental animal, and acute necrosis of the pancreas resulted. Lombardi, in discussing the subject of pancreatic juice activation as a factor in the production of acute pancreatic necrosis, states that the enzymes are known to be activated by certain substances, such as enterokinase, a secretion of the upper intestinal tract. He also believes that there is a cytokinase, such as may be liberated by leucocytes and perhaps other cells, and a bacteriokynase. Relative to the latter, it is difficult to determine whether this activating substance is a product of bacterial lysis or a toxin which may be elaborated by the living bacteria. The conclusions of Lombardi are sustained by clinical experience, for it is known that

operations can be performed upon the pancreas without subsequent necrosis if three precepts are observed: sharp dissection, complete hemostasis, and no infection.

It would appear that bacteria play a rôle in the experimental production of pathologic changes in the gallbladder with the use of pancreatic juice. It is a known fact that gallbladder disease cannot be produced by the introduction of bacteria into the gallbladder, but it has been conclusively proved that pancreatic juice, with bacteria, leads to varying degrees of changes in the wall of the gallbladder. Unactivated, sterile pancreatic juice is practically inert, for Dragstadt has shown that 100 c.c. of pancreatic juice which has been passed through a Berkefeld filter can be introduced into the peritoneal cavity of a dog and cause no disturbance or fat necrosis, but 50 c.c., or less, when not passed through a Berkfeld filter, will produce death. On the other hand, bacteria are commonly present in the biliary and pancreatic secretions. It is known that, in the dog, in 70 per cent of cases, the mucosa of the gallbladder contains bacteria, and in 30 per cent, the bile contains colon bacilli and staphylococci; moreover, Dragstadt reports that in 90 per cent of experimental animals, the pancreas yields positive cultures. Considering the fact that the dietary habits of the human are not those of the dog, nevertheless, it is reasonable to conclude that frequently in the human being the bile and pancreatic secretion will contain bacteria.

The preponderance of the experimental evidence indicates that stasis is an important factor in the production of gallbladder disturbance when pancreatic juice is mixed with the bile. In the human being, stasis is relatively common. It may be a purely functional affair or produced by disease of the papilla or due to intermittent occlusion at the papilla by stone. I am of the opinion that low grade stasis, such as may be produced by spasm or partial stenosis at the papilla, is more likely to produce a pancreatic juice reflux into the gallbladder than stone impaction in the ampulla. The probability of an impaction of a small stone in the papilla, thus creating a continuous pathway, requires a high union of the two ducts so that the stone will not interfere with the passageway between the biliary and pancreatic ducts. This may be relatively uncommon. Birgfeld cites a case in which, after a gastric resection, the patient developed an acute biliary peritonitis. At operation the duodenal stump was found to be well closed, but the gallbladder was markedly distended and dark greenish brown in color. He attributed the change in the gallbladder to a reflux of duodenal contents into the biliary passages, since later he demonstrated in this individual by roentgen study that the barium entered the duodenum and distended it. A man upon whom I had performed a posterior gastroenterostomy for a pyloric carcinoma died approximately twelve days after the operation. The autopsy disclosed a gangrenous cholecystitis. It is entirely

possible that in these cases a reflux of pancreatic juice may have been the etiologic factor in the production of the gallbladder pathology.

HYPOTHESIS

Embryologic and anatomic evidence indicates that it is possible, in the human, for a continuous pathway to exist between the pancreatic and biliary systems in a considerable percentage of cases. Since the secretory pressure of the pancreas is greater than that of the biliary system, in the presence of obstruction at the papilla, it is possible for pancreatic secretions to mix with bile in the common duct. The obstruction may be complete, such as that produced by a stone impacted in the ampulla; or it may be incomplete or intermittent. Ivy has shown that biliary stasis is common without definite organic obstruction of the duct at its entrance into the duodenum. There are many clinical examples proving the fact that the pancreatic juice may enter the gallbladder and that, associated with this phenomenon, there may exist necrosis of the gallbladder with or without a biliary peritonitis. A number of observers have called attention to cases of acute gangrenous cholecystitis in which cultures from the gallbladder walls or contents were sterile, the process suggesting an acute digestion of the gallbladder wall. Recently Colp, Gerber, and Doubilet reported three cases of acute cholecystitis, the gallbladder bile in two containing both amylase and trypsin and in one containing only amylase. In two cases, cultures were negative, the other revealing both *Bacillus Friedländer* and *Bacillus coli*. In two cases, free bile was present in the peritoneal cavity, and in one case, fat necrosis was present throughout the gallbladder wall and in the omentum adherent to the gallbladder. Experimental evidence clearly indicates that the pancreatic juice may affect the walls of the gallbladder under variable conditions and produce different types of changes, varying from acute necrosis to chronic hyperplastic and inflammatory reactions. Assuming these observations to be true, the following hypothesis may be constructed:

Under normal conditions the bile passes through the ducts to be emptied into the duodenum, some entering the gallbladder to be concentrated and later to be expelled into the common duct. The pancreatic juice may enter the duodenum by a direct passage or may fuse with the bile in the ampulla. Brackertz has shown experimentally that even in the presence of extensive changes in the gallbladder wall, due to the presence of pancreatic juice the bile ducts are not involved. He believes this is due to the fact that there is much more elastic tissue underlying the mucosa of the ducts than is in the wall of the gallbladder. The elastic tissue is very resistant to the action of pancreatic juice. In experiments in which he introduced pancreatic extract mixed with colon bacilli into the common duct and obstructed

the duct, marked changes took place in the wall of the gallbladder, but the ducts remained normal except for, at times, localized necrotic changes in the wall of the duct at the site of the duct puncture. It is therefore logical to assume that without stasis and with normal anatomic and physiologic mechanisms even though the duct bile may be mixed with pancreatic juice no pathologic changes take place in the biliary passages.

With low grade stasis in the biliary passages such as may be produced by disturbed papillary function or by complete or incomplete obstruction at the duodenal end of the biliary and pancreatic ducts, the bile mixed with pancreatic juice may remain in the ducts for a considerable time, the bile-pancreatic juice ratio being altered, and variable amounts of pancreatic juice enter the gallbladder. The disturbance created in the gallbladder will then depend upon known and unknown factors. If the pancreatic juice content is low and no bacterial contamination is present, no changes may occur in the wall of the gallbladder. If, however, the pancreatic juice content is higher, the stasis prolonged, and possibly a low grade bacterial contamination is present, changes may occur as described by Andrews, Goff, and Hrdina. They have shown with the introduction of pancreatic juice into the gallbladder of the dog that the cholesterol content of the bile is not altered, but that the concentration of the bile salts is reduced to less than one third. In five experments, with unfiltered pancreatic juice, all the bile salts had been absorbed leaving the cholesterol precipitated. Therefore, in the human being, the low dilution stage may be a factor in the production of gallstones. When the concentration of pancreatic enzymes in the gallbladder bile is high, the pathologic changes produced will depend upon complete or incomplete activation of the pancreatic enzymes in greater or lesser dilution in contact for short or long periods of time, sufficient time and concentration being necessary for necrosis.

It is not the purpose of this paper to convey the idea that all cases of cholecystitis or gallstones are produced by a reflux of pancreatic juice into the gallbladder; however, I am convinced that the cause of selected cases of acute necrosis and acute gangrenous cholecystitis and also cases of chronic cholecystitis with or without stone can be found in a reflux of pancreatic juice into the gallbladder.

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THE EQUAL DIVISION AND DISTRIBUTION OF THE DIET AND INSULIN IN TREATING THE DIABETIC WITH SURGICAL COMPLICATIONS AND ACUTE INFECTIONS*

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A NEED exists for an outline of a simple, practical, and effective treatment for diabetic patients having acute infections and surgical complications.

Much has been written on the various types of diet for these patients, but little has been said of its distribution, or of that of the insulin. This results in confusion to all but specialists in the treatment of diabetes. Clarke and others[1] reported benefits from equal division of the insulin with frequent administrations, but apparently the diet was not so altered.

The prescribing of the total diet may be dispensed with by noting that the average adult for each twenty-four hours requires a minimum of ⅔ gm. protein per kilogram of body weight, and more in prolonged fevers. The carbohydrate allowance is at least 150 gm. and preferably 200 or higher in the overweight patient. The fat quota is low but is sufficient to keep the total calories at 25 per kilogram of body weight or higher in prolonged fevers.

It is sufficient to say that all diabetics with acute infections, fever, or those requiring surgery should receive unmodified insulin temporarily at least.

DIET AND INSULIN DISTRIBUTION

In dealing with acute infections and postoperative and postketotic diabetic patients and recently during confinement of our pregnancy cases, we have adopted the practice of dividing the diet—liquid, soft, or combined as indicated—into four, five, or six equal feedings according to the intensity of the fever, the magnitude of the operation, the severity of the diabetes and the necessity of having these feedings equally spaced in the twenty-four hours. Similarly the insulin is equally divided so that each dose precedes or, in the case of digestive upsets, may follow each feeding. This regime has been illustrated in a previous publication.[2]

Following operations, for six to twenty-four hours, when oral feeding may be impractical, or at other times when the patient is unable to take food by mouth, the carbohydrate equivalent of a feeding may be given

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intravenously in the form of glucose, 5 or 10 per cent solution, at the scheduled feeding times. In this event, two-thirds of the planned dose of insulin is given.

In presenting the basis for this treatment, a word about the effects which changes in the total metabolism have on diabetes is appropriate, because these changes indicate the alteration of the program from that for the noncomplicated to that for the complicated diabetic. Allen, and his associates,[3] proved that a reduction in body weight, as a result of undernutrition, improved the diabetic's food tolerance and that an increase in the body weight had the opposite effect. It is well known that if a diabetic, standardized on a diet and insulin regime, gains in weight, the insulin need is increased. This is especially noticeable in children. Hyperthyroidism,[4-6] adrenal[7] and certain pituitary tumors also aggravate the diabetes and in consequence increase the insulin requirement. In the event of pregnancy,[8] infection,[9] fever, and toxemia (see illustrative cases below), desiccated thyroid,[2, 10] epinephrine, dinitrophenol or prolonged roentgen ray therapy, increased caloric intake, and some cases of acute myocardial insufficiency, the same result ensues. In other words, *any factor, physical exercise excepted, which increases the total metabolism, increases the insulin need.* The previous dosage becomes inadequate. The reverse is also true. Removal of the thyroid gland in man, as shown by Wilder,[11] and Rudy,[12] or the anterior pituitary or adrenals as proved experimentally by Houssay and Biasotti,[13] Long and Lukens,[14] and others, lessens the severity but does not cure the diabetes; so also does a reduction in weight (Benedict[15] found that a loss of 10 per cent of the body weight decreased the total metabolism 19 per cent), a reduction in the caloric intake, eradication of infection or toxemia, stopping desiccated thyroid,[5, 10, 16] epinephrine or dinitrophenol therapy, giving iodides to diabetics with Graves' disease,[14, 18] the spontaneous development of myxedema,[11] and approaching old age.* Though altering body physiology in divergent ways, these conditions in common *reduce the total metabolism and simultaneously alleviate the diabetes.* Exception is made to the cessation of physical exercise, which, while decreasing the total metabolism, does not alleviate the diabetes, but has rather the opposite effect. A relative and absolute increase in combustion of carbohydrate coincident with physical work done may account for the decrease in the blood sugar which accompanies the increased total metabolism associated with physical exercise and vice versa.

The total metabolism must not be confused with the basal metabolism. The total metabolism is elevated if the basal rate is above normal, but the basal metabolism may be normal with an abnormally high total metabolism. For instance, the basal rate may be normal for the surface

*The decrease in the total metabolism in old age accounts, we believe, for the remarkably few new cases of diabetes in people over sixty-five years of age.

area of a patient weighing 120 kg. but whose normal weight is 80 kg. It is obvious, however, that the total metabolism is above that of the normal person of the same age, height, and sex.

Clinically, increases in the total metabolism, that due to physical exercise excepted, shorten the period of insulin effectiveness. The dose of insulin which previously maintained a satisfactory blood sugar concentration no longer does so, hence the need for larger doses or an approximation, and an increased number of doses is created.

The uncomplicated diabetic exercises in the daytime. It is physiologic that the diet and insulin should be given at this time when the metabolic activity is greatest.

The diabetic with infection or fever confined in bed—thus deprived of the influence which exercise has on the oxidation of sugar—has an increased total metabolism throughout the twenty-four hours, hence *the need for food and insulin is equally as great at night as in the daytime.* These needs are best supplied with least difficulty and, to our belief, with best results by frequent and equal spacing of insulin and meals. Illustrative cases as well as a summary of eighty cases are presented. By virtue of this equal division and spacing of the diet, the insulin can be divided into equal doses. The insulin, based on the results of fractional urinalyses, a specimen before each feeding being examined, is increased to overcome glycosuria and hyperglycemia, or decreased when the need arises to prevent insulin reactions.

Six feedings and six doses of insulin are used in the presence of severe infections, high fever, and immediately after surgical operations (four hourly feedings). The less severe infections with moderate fever or a moderating severe infection would indicate a division into five meals and five doses of insulin given at five-hour intervals, and a mild fever or a mild infection, four meals and four doses of insulin given at six-hour intervals. Three meals and three doses are resumed when convalescence is established.

ILLUSTRATIVE CASES

CASE 1.—F. H., female, aged sixty-seven years, was admitted to the Pennsylvania Hospital on March 2, 1934, for the treatment of diabetes and gangrene. She complained of severe pains in her legs. Her family history was unimportant, and her only past illness was pneumonia in childhood.

Present Illness: In 1931, after a minor injury to her right great toe, a vesicle appeared at the site of injury. The entire toe became inflamed. Glycosuria was found, and a diagnosis of diabetes with gangrene was made. A special diet and insulin were given, and the patient was confined to bed for five weeks with good results.

Five days prior to admission, another vesicle appeared on the fifth toe of the left foot. The underlying tissues became black. At this time, an ulcer appeared on the distal third of the right leg.

The positive findings on physical examination were: undernutrition, marked thickening of the peripheral arteries, arterial hypertension, retraction of the left

nipple with an irregular mass, which proved to be a benign tumor in the left upper quadrant of the breast, emphysema with fine crackling râles at the base of each lung, and mitral insufficiency with enlargement of the heart. There was a feeble pulsation in both dorsalis pedis arteries, and the feet were cool to touch. The affected toe was gangrenous, emitting a foul odor and a purulent discharge.

The patient was considered a poor surgical risk. Watchful waiting was advised by the surgeons.

Insulin was begun again on May 4 and was steadily increased. In May and June, the gangrene extended, involving the plantar surface of the foot with profuse purulent discharge. There was continued fever. Radical amputation was decided upon.

Control of the diabetes was maintained by adding to the insulin, which was given in five doses, and with a diet of 70 gm. of protein, 36 gm. of fat, and 200 gm. of carbohydrate (1,400 calories) (see Table I). The diet was given in five equal feedings evenly distributed throughout the twenty-four hours (five hourly intervals).

On the day of operation, June 6, the usual amounts of insulin were given before liquid meals at 7:00 A.M. and 10:00 A.M. At 11:45 A.M. a spinal anesthetic (neocaine) was administered, and at midday 10 units of insulin were given. The left leg was then amputated at the midthigh level by Dr. A. Walkling. Following the operation 250 c.c. of a 10 per cent solution of glucose was given intravenously. This was repeated at 4:00 P.M. with 20 units of insulin. Ginger ale and orange juice were given freely.

The liquid meals, 6, equally spaced, in twenty-four hours, were resumed at 8:00 P.M., each being preceded by 22 units of insulin. House diet of appropriate values was allowed on June 8. A rapid decrease in the insulin need ensued. On June 13, the feedings and insulin doses were reduced to 5 in number, and on June 15, to 4. Uneventful recovery ensued.

CASE 2.—H. M., male, insurance salesman, aged 29 years, weight 66 kg. (146 pounds), entered the Pennsylvania Hospital January 1, 1936, complaining of pain in back, made worse by deep breathing, fever, and general weakness.

Family History: Irrelevant. Past Illnesses: Influenza in 1918, furuncle in groin in 1920, and the diabetes recognized in 1932. This was controlled by a weighed diet with insulin 3 times daily. The dosage at the onset of the present illness was 20 units before breakfast, 12 before lunch, and 14 in the evening.

Present Illness: On December 15, he contracted an acute upper respiratory tract infection. He continued at work with no improvement and was seen by one of us (G. G. D.) on December 27. He had a nasal discharge and a productive cough but no fever. On January 1, he was awakened by a sharp, stabbing pain in the left side of his back. A productive mucopurulent and increasingly blood-streaked sputum followed, and he had a severe chill. He became nauseated, and because of this omitted his noon insulin. The evening nourishment and insulin were taken. He was then seen by Dr. C. Nissler who, finding early signs of lobar pneumonia, advised his admission.

Physical Examination: Appeared acutely and seriously ill, temperature, 104° F.; pulse, 104; respirations, 28; blood pressure, 120/60; moderate cyanosis of mucous membranes; face flushed, tongue coated, dental caries, nasal discharge, injection of nasopharynx, limited expansion with dullness to percussion, and moderate suppression of breath sounds over the right lower lobe. The liver edge was palpable.

Progress Notes: On January 2, the signs of consolidation were pronounced. The patient continued very ill, and on January 6, signs of fluid at the right base were observed, and 500 c.c. of serosanguineous fluid were removed. On January 7, he was quite jaundiced, and signs of consolidation of the entire right upper lobe

TABLE 1*

DIET, INSULIN TREATMENT, AND LABORATORY DATA OF A DIABETIC PATIENT WITH GANGRENE NECESSITATING AMPUTATION. CASE 1

DATE 1934	TIME	DIET				INSULIN						BLOOD SUGAR	GLYCOSURIA				
		P	F	C	CAL.												
June 4		70	36	200	1400	26	24	25	25	23			0	0	0	0	
5		70	36	200	1400	26	24	25	25	23			0	0	0	0	
6	7:00	12	7	33	243	26						98					Liquid diet
	10:00	12	7	33	243	24											
	11:57					10											
	12:20																Leg amputated
	12:35			25	100												Glucose by vein
	4:00			60	240	20						67	Negative				
	8:00	12	7	33	243	22							Negative				
	12:00	12	7	33	243	15											
7	4:00	12	7	33	243	15						130					
	8:00	12	7	33	243	15							Negative				
	12:00	12	7	33	243	15											
	4:00	12	7	33	243	15							Negative				
	8:00	12	7	33	243	15											
	12:00	12	7	33	243	15											
8		70	36	200	1400	12	12	12	12	12	12	122	0	0	0	0	House diet (6 meals)
9		70	36	200	1400	9	9	9	9	9	9	121	0	0	0	0	
10		70	36	200	1400	7	7	7	7	7	7	133	0	0	1	0	
11		70	36	200	1400	6	6	6	6	6	6		0	0	0	0	
12		70	36	200	1400	6	6	6	6	6	6	126	0	0	0	0	
13		70	36	200	1400	6	6	6	6	6		181	0	0	0	0	5 meals
14		70	36	200	1400	6	6	6	6	6		133	0	0	0	0	
15		70	36	200	1400	9	9	9	9			152	0	0	0	0	4 meals

*Illustrates the equal division of the diet into 6, then 5, and lastly 4 feedings as convalescence was established. The insulin was similarly divided and likewise equally spaced in time of administration.

TABLE II*

DIABETES COMPLICATED BY LOBAR PNEUMONIA, CASE 2

DATE	DIET				INSULIN		BLOOD SUGAR		URINE SUGAR		
	P	F	C	CAL.	DISTRIBUTION	TOTAL UNITS	A.M.	P.M.	QUAL.	QUANT. GM.	
12/27-31	100	126	115	2000	**27-13-19**	59					Prior to admission
1/ 1	60	40	250	1600	**27- 0-19-10-10-10**	76					Admitted
2	60	40	250	1600	**10-10-10-10-10-10**	60	280		4+	5.7	6 feedings
3	60	40	250	1600	**10-10-10-10-10-15**	65	283		4+		
4	60	40	250	1600	**15-15-20-20-20-20-***10*	120	293	326	4+ to 2+	23.1	
5	60	40	250	1600	**50-***10*-**40-***10*-**50-***10*-**60-***20*-**50-***20*-**50**	370	307	150	4+ to 0	34.7	
6	60	40	250	1600	*10*-**30-30-***10*-**30-***10*-**30-***10*-**35-***10*-**35**	240	223		1+ to 4+	18.0	
7	60	40	250	1600	*10*-**35-***10*-**35-***10*-**35-***10*-**35-***10*-**35-***10*-**35**	270	205		1+ to 2+		
8	60	40	250	1600	*10*-**35-***10*-**35-***10*-**35-***10*-**35-***10*-**35-***10*-**35**	270	180		2+ to 0		
9	60	40	250	1600	*10*-**35-***10*-**35-35-***10*-**35-35-35**	240	66		0		
10	60	40	250	1600	**35-35-35-35-30-35**	205	123		0 to 1+		
11	60	40	250	1600	**30-30-30-30-30**	150	55		0 to 1+		5 feedings
12	60	40	250	1600	**25-25-25-25**	100	185		0		4 feedings
13	60	40	250	1600	**20-20-20-20**	80			1+ to 3+		
14	60	40	250	1600	**20-20-25-25**	90	249		0 to 1+		
16	60	40	250	1600	**25-25-25-25**	100	98		0 to 1+		
19	60	40	250	1600	**20-20-20-20**	80	93		0 to 2+		
20	60	40	250	1600	**20-12-19- 8** (9 P.M.)	59			1+		3 feedings with late nourishment
2/ 1/32	60	40	250	1600	**23-12-17- 6**	58	91		0		
4	60	40	250	1600	**23-12-23†**	58			0 to 1+		†Protamine insulinate
9	60	40	250	1600	**23-12-23†**	58	150				

Heavy figures represent dose prior to nourishment, italics those between feedings—see text.

*Depicts the diet and insulin and their distribution, and laboratory data.

TABLE III

DISORDERS COMPLICATING DIABETES WITH ILLUSTRATIVE DIET AND INSULIN REGIMES EMPLOYED

	NO. OF CASES	DIET					INSULIN		DEATHS
		P	F	C	CAL.	FEEDINGS	HIGHEST TOTAL	NO. OF DOSES	
Acute abdominal conditions: abdominal teratoma (1), appendectomy (4), cholecystitis and cholecystectomy (4), hysterectomy (2), pancreatitis (subacute) (1), salpingectomy (bilateral), right oophorectomy, removal of cyst from broad ligament (1), suspension of uterus and perineal repair (1)	14	60	17	225	1300	6	130	6	0
Amputations	9	70	35	200	1400	6	123	6	2
Carcinoma (penis, cervix, thyroid, and esophagus). (Two recovered from the operation but died later of metastases.)	4	60	51	225	1600	6	116	6	1
Coma-impending or after coma only.	8	80	42	200	1500	6	120	6	0
Cutaneous and subcutaneous infections: abscesses (5), carbuncles (9) (one case had complicating grave cardiac decompensation), cellulitis and erysipelas (1), furunculosis, large abscess of scalp and pneumonia (1) (died on third day after admission)	16	65	60	200	1600	6	240	6	1
Gangrene with secondary infection, but without amputation	3	65	42	190	1400	5	85	5	0
Gonorrheal epididymitis	1	70	82	120	1500	4	80	4	0
Mastoidectomy operation	2	65	37	200	1400	6	72	6	0
Osteomyelitis (one patient died 6 months after operation of pulmonary tuberculosis)	4	60	73	150	1500	4	82	4	0
Pneumonia (lobar)	11	60	62	225	1700	6	98	6	1
Postpneumonic empyema (thoracotomy)	2	75	66	200	1700	5	75	5	0
Postpneumonic lung abscess (patient whose regime is illustrated had Friedländer's pneumonia with bilateral pulmonary abscess, 1 drained by posture, and 1 by operation. Recovery)	2	50	55	200	1495	6	175	6	0
Pneumonia (broncho-) (one patient was in precoma, and had an acute pyelitis; the other had a complicating gangrenous foot; both elderly)	3	60	62	200	1600	5	125	5	2
Trauma from auto accident and fractured tibia	1	75	120	200	2180	5	60	5	0
Total	80								7

with beginning resolution at the base were noted. One hundred cubic centimeters of serous fluid were removed. Clinical improvement with a fall in temperature by lysis, leading to recovery, began on January 8.

Laboratory Studies: Studies pertaining to the diabetes are presented in Table II. The urinalysis (on admission) was normal except for the presence of clumps of white blood cells. Blood Count: January 2, hgb. 77 per cent; R.B.C., 3,920,000; W.B.C., 10,350. January 3, 15,300; January 12, 16,600. The blood Wassermann reaction and the Kahn precipitation tests were negative.

Sputum: Culture and routine typing, pneumonia Type I—pathogenic to mice. Pleural fluid: Smear revealed many polymorphonuclear cells, few lymphocytes, frequent large mononuclears and some large multinucleated cells and no organisms. There was no growth on culture. Blood culture—no growth (twelve days). Roentgen ray studies by Dr. Paul Bishop confirmed the physical findings.

Comments.—The diet was 60 gm. protein, 250 gm. carbohydrate, and sufficient fat to make 1,600 calories and was given in liquid form and divided into 6 equal feedings, equally spaced in the twenty-four hours. *Insulin,* 10 units were given before each of the 6 feedings, but this proved inadequate and the dosage was rapidly increased to 50 units before each nourishment. This dosage proved insufficient. The effect of each dose of insulin was exhausted before the next one was due. Accordingly at the midpoint between feedings, an additional 10 units were given. This is the only patient, in our experience, in whom this additional measure has been found necessary. The unusually severe demand resulted in a total dosage of 370 units on January 5.

The midpoint doses (between meals) were discontinued on January 10, and the doses before nourishments were gradually decreased (see chart). The diet was divided into 5 feedings on January 11, and on January 12 into 4 feedings with 25 units of insulin before each. Later, 20-unit doses were sufficient. On January 20, the diet was divided into 3 meals with a bedtime nourishment saved from supper with 4 doses of insulin, 20 units before breakfast, 12 at noon, 19 before supper, and 8 at 9 P.M.

By using protamine insulinate (Lilly), the two evening doses were combined and given before supper. Details of this aspect of the study will be given elsewhere.

Table III depicts: (1) the results in treating diabetics having acute infections and surgical complications; (2) an illustrative diet and its distribution for each group; and (3) an illustrative insulin dosage and its distribution. Less severe disorders (tonsillitis, peritonsillar abscess, tonsillectomies, furuncles, influenza, and la grippe), from which there were no deaths but for which the same treatment was used, have been omitted, as have four diabetics—with pyonephrosis, apoplexy and pulmonary tuberculosis with ketosis, carbuncle and septicemia with ketosis, erysipelas with an extensive subcutaneous abscess of the scalp and generalized pyodermia, either moribund or hopelessly ill before this treatment was begun.

Of the 80 recorded cases, there were 7 deaths (8.7 per cent). One of these was due to lobar pneumonia, the only fatality in 11 consecutive cases (9 per cent mortality). While the pneumonia mortality rate is low, the frequency of complications—pleural effusion, empyema, and lung abscess—was higher than in nondiabetics.

Two deaths following major amputations were attributable to uremia and bronchopneumonia, and septicemia. One patient died following gastrostomy for carcinoma of the esophagus. At autopsy, an esophageal bronchial fistula was also found. One death occurred from hypostatic pneumonia following a series of furuncles and a large abscess of the scalp. The remaining two deaths were due to bronchopneumonia, uremia and gangrene, and bronchopneumonia, acute pyelitis, and ketosis.

SUMMARY AND CONCLUSION

Equal division and spacing of the diet and insulin is advocated for diabetic patients during the postoperative period, during acute infections, and for two or three days following attacks of diabetic coma because:

1. Of the simplicity of the plan and the ease with which the diabetes can be controlled.

2. There is less tendency to great fluctuation in the blood sugar, less tendency to ketosis, and less tendency to hypoglycemia.

3. Our results on this regime are in happy contrast to those when longer periods without food and insulin were allowed. Furthermore, we believe the results, the time factor, and the number of patients thus treated amply justify the treatment employed.

The slow absorption, the prolonged effect and the stabilizing influence that protamine insulinate has on the blood sugar were shown to advantage when this preparation was combined with the regular insulin treatment during convalescence.

The authors gratefully acknowledge the kind cooperation of Drs. C. Mitchell and W. E. Lee, the late E. Klopp, the late Thomas McCrae, John Gibbon, Sr., and John Flick in permitting this study to be made on their patients at the Jefferson and Pennsylvania Hospitals.

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PERFORATION OF THE GALLBLADDER

Analysis of Forty-Six Cases*

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THIS series of 46 perforations of the gallbladder which occurred in 886 operations for cholecystic disease, a percentage of 5.2, is being reported as a further argument for recognition of the high incidence of the complication and the urgent necessity for operation before its development.

In 1934, Eliason and McLaughlin gave a tabular review of cases of perforated gallbladder as published by several authors to that time, including figures of incidence and mortality. They showed that the incidence ranged from 0.9 to 2.5 per cent. The average mortality in cases of five authors whom they quoted was approximately 47 per cent. In addition, they reported their own series of 9 perforations found in 500 cases of gallbladder disease, an incidence of 1.8 per cent, with one death, or a mortality of 11 per cent.

Eight of the 46 patients in the group reported here died from complications having some bearing upon the operation, constituting a mortality of 17.4 per cent. In the 834 nonperforated cases there were, in addition, 2 probable perforations, 3 partial perforations, 25 in which the gallbladder was gangrenous, 28 in which pericholecystitis was a complication, and 9 in which local peritonitis had developed.

Of the total number of patients, 621 were women, and 265 were men; whereas, 22 of those whose gallbladder had perforated were women, and 24 were men. From this, it is apparent that the ratio of perforations is much higher in men. A similar finding has been reported by others, but no explanation has been offered for this peculiar circumstance.

Table I

Perforation of the Gallbladder: Incidence and Mortality

	No. Perforations	Incidence	Deaths	Mortality
6,800 cases—five authors (quoted by Eliason and McLaughlin)	96	0.9 to 2.5%	-	42 to 52%
500 cases—reported by Eliason and McLaughlin	9	1.8%	1	11.0%
886 cases—reported by R. L. Sanders	46	5.2%	8	17.4%

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The average age of the men in whom perforation had taken place was 55 years, the oldest having been 73 years, the youngest 34 years. The ages of the women averaged 50 years; the oldest was 70 years, the youngest 23 years. The average age of the patients who died was 60 years.

The perforations of the gallbladder in this group are classified as (1) chronic, (2) subacute, and (3) acute according to the severity of symptoms, the activity of the process, and the duration of the present attack. In the infections regarded as chronic, recovery had taken place, all the local infection had been controlled, and the perforation had been well protected. Practically all the patients were ambulatory, and the operation could be classified as elective. The subacute cases were those in which the violence of the symptoms had been spent, the infection was abating, and the walling-off process was well under way, but in which it was evident from the course of the disease that the patients would not completely recover. The duration of the attacks in this group varied from a few days to two or three weeks. In the acute cases, the perforation had occurred recently and the infection was active, the time which had elapsed from the onset of symptoms to the operation having ranged from a few hours to three or four days.

The perforation was regarded as chronic in 13 cases, subacute in 11, and acute in 22. Of the total number of patients with chronic, subacute, and acute perforations, 22 had empyema of the gallbladder; frank pus was found in the gallbladder of another; in 9 patients the gallbladder was gangrenous; a marked pericholecystitis was present in 1, local peritonitis in 1, a spreading peritonitis in 2; and in 1, an intraperitoneal hemorrhage had taken place. In none of the patients had the intestinal tract been sufficiently involved to produce obstruction.

Stones, which were present in 42 of the 46 cases, were distributed as follows: gallbladder alone, 23; ducts alone, 6; and gallbladder and ducts, 13.

Cholecystectomy was performed in 36, partial cholecystectomy in 4, and cholecystostomy in 6 cases. Two of the patients on whom a cholecystectomy was done had previously had a cholecystostomy.

In addition to the gallbladder operation, one or more surgical procedures were carried out in 32 cases, in 5 of which 3 operations were performed. The common duct was opened in 11 cases, and the appendix was removed in 21. A separate McBurney incision was made in 4.

Nine of the 13 chronic perforations had entered the liver, producing localized abscesses. In one case, the opening was in the fundus and was protected by adhesions. In another, the gallbladder had perforated onto the stomach; this, also, was guarded by adhesions. Two

of the patients with chronic perforations developed complications which brought about death. One of these had a perforation into the duodenum, with adhesions about the fistula; a cholecystectomy and choledochotomy were done and the fistula repaired. The remaining patient had a perforation onto the stomach, which was protected by adhesions. The operation consisted of a partial cholecystectomy and choledochotomy.

Of the 11 subacute perforations, 7 had opened into the liver, 2 were protected by adhesions, and 1 had taken place into the abdominal wall, with subsequent rupture of the wall and drainage of stones to the surface. Another perforation of this group had entered the stomach and duodenum, forming two distinct fistulas, representing a spontaneous cholecystogastrostomy and cholecystoduodenostomy. Five of these patients had empyema with protected perforation. Stones were found in the gallbladder in all the subacute cases, and in the ducts in 5 cases. A choledochotomy, with removal of stones, was done in 3 of these.

Cholecystectomy was performed in 9 of the subacute cases, partial cholecystectomy in 1, and cholecystostomy in 1. In the case of perforation into the stomach and duodenum, it was necessary to do a partial resection of the stomach and remove the gallbladder at the same time, from which the patient failed to recover.

The perforation was regarded as acute in 22 cases. Eight of these were definitely protected by omentum, 3 had taken place into the liver, and 1 into the hepatic flexure of the colon; 2 had occurred at the juncture of the cystic duct and were protected by recent inflammatory tissue; 1 had perforated onto the duodenum; 3 had entered the general peritoneal cavity; 1 ruptured during manipulation while separating adhesions preparatory to removal of the gallbladder; and in 1 case there was a spontaneous separation of the gallbladder from its liver attachment and sloughing of the duct, permitting the gallbladder to be lifted out without effort. The blood supply was sufficiently

TABLE II

CLASSIFICATIONS

Type:	
Chronic	13
Subacute	11
Acute	22
Stones:	
Gallbladder alone	23
Ducts alone	6
Gallbladder and ducts	13
Operation:	
Cholecystectomy	36
Partial cholecystectomy	4
Cholecystostomy	6

damaged in 9 cases to produce gangrene. Stones were present in the gallbladder in 14, and in the ducts in 9. The common duct was opened in only 2 cases.

The gallbladder was removed in 15 of the acute cases, a partial cholecystectomy was done in 2, and a cholecystostomy in 5. Five of the patients in this group died, making a total mortality of 22.7 per cent in the acute cases. It is obvious from this that operation was postponed too long after the onset of symptoms; earlier surgical intervention would have saved a greater number of these patients.

The symptoms of the 46 patients had persisted for varying periods, from a few days to thirty-five years. A history of previous symptoms of cholecystitis was obtained from 32 patients. Twenty-nine gave pain as the chief symptom in early attacks, and 15 of these had noted that it radiated to the scapular region; in 3, the pain was described as a hunger sensation. Five patients had had attacks of violent nausea and vomiting, while 8 had been bothered with gaseous distention. Chills, fever, and damage to the vascular system had occurred in several. Anorexia, constipation, and a mild jaundice had been noted in former attacks by one-fourth the group. Only 4 patients were seen in the first attack. One of these had been sick for a week, another for two weeks, and the remaining 2 for four and five weeks, respectively, before coming to surgery.

Many of the patients stated that the present attack was acute at the onset, became less severe for a day or two, then was exacerbated as peritonitis developed. In 35 cases, the average duration of the attack for which the operation was performed was two to three weeks. The symptoms had been present for twelve hours in the shortest, and in the longest, they had persisted, with alternating periods of mild discomfort, for six months. In the majority of cases of a prolonged history of the present attack, the perforation had, of course, become chronic. In a few, however, an acute perforation followed attacks lasting for several weeks. Multiple ruptures were found in 5 cases, the primary opening having been protected by adhesions and newly formed tissue.

Physical examination revealed that epigastric tenderness and muscular rigidity were present in practically every case and were exaggerated in those in which fairly extensive local peritonitis had developed. A definite mass was palpated in 15, and in the remainder there was thickening of the tissues. A mild or moderate leucocytosis was recorded in all the cases in which the blood cell count was made.

A correct preoperative diagnosis was made in 4 of the acute cases and was suspected in a few others. Cholecystitis, or empyema of the gallbladder, was the preoperative diagnosis in 35, obstructing duodenal ulcer with cholecystitis in 1, perforating duodenal ulcer in 1,

abscess of the abdominal wall secondary to gallbladder perforation in 1, and acutely inflammatory, high-lying appendix in 1. Three of the patients were submitted to exploration on an indeterminate diagnosis.

Only 14 of the entire number of patients with perforation had a roentgenologic study; 6 of these had a functionless gallbladder, the others, varying degrees of impairment. The diagnosis was sufficiently clear without the aid of a roentgenogram in the majority of cases.

The average duration of hospitalization of the 46 patients was 22 days; the briefest period was 10, the longest, 60 days.

In this series, there were 8 deaths which occurred following operation. In one other case, the wound had healed, bile was passing normally through the bowel, and the patient had recovered entirely from a cholecystectomy and choledochotomy. He was 71 years of age, had previously undergone a prostatic resection, and was handicapped by a damaged myocardium. A flare-up of the urinary tract infection weakened his condition and was responsible for his death, twenty-one days after operation.

A résumé of each of the 8 fatalities follows:

CASE 1.—A woman, aged 70 years, had been troubled with stomach disorder for thirty-five years, yet had never had an attack of acute colic. Recently her complaint had been a periodic burning, heavy sensation in the stomach and epigastrium, which began a few hours after meals and was relieved by the ingestion of food. She had not been jaundiced. The present attack was abrupt in onset, with severe pain, nausea, and vomiting as the chief symptoms. Twenty-four hours later her temperature was slightly elevated, and the right upper abdominal quadrant was tender and rigid; no mass could be palpated. The preoperative diagnosis was a probable perforated duodenal ulcer.

Exploration revealed a large, thick-walled gallbladder containing purulent material and many stones. A perforation was found near the liver border but was protected by omentum, which formed the wall of a fairly large abscess. The liver was congested and extended down four fingerbreadths below the costal margin. The appendix was subacutely inflamed and was removed. Cholecystectomy was performed, free drainage instituted, and the abdominal wall closed. The patient's condition was satisfactory for three days; both lungs then became congested, her temperature rose, delirium and disorientation followed, and she died on the fifth day postoperatively from bronchopneumonia and myocardial insufficiency.

CASE 2.—A woman, aged 65 years, gave a definite history of gallbladder disease covering a period of thirty years. Her distress had been more acute during the previous three years. Four weeks prior to the operation, she had had an attack of severe pain, nausea, vomiting, and fever, which had failed to subside completely. She had also lost weight and strength.

The patient's temperature was slightly elevated, and her blood cell count showed an increase in leucocytes. A large, tender mass was palpated in the gallbladder region, and a moderate muscular rigidity indicated local peritonitis. The preoperative diagnosis was acute empyema of the gallbladder with perforation.

At operation, the gallbladder was found to have perforated into the hepatic flexure of the colon, forming a cholecystocolic fistula. The gallbladder contained one large stone and purulent material. The liver border and contiguous tissues

were acutely inflamed; local peritonitis was extending; and a considerable quantity of infected free fluid filled the right upper quadrant of the abdomen.

The gallbladder was removed, the colon fistula repaired, free drainage established, and the abdomen closed. A blood transfusion was given at once. Following operation, there was a profuse and malodorous drainage, but it was apparent that much of the infectious material was being absorbed. The patient declined gradually and died ten days after operation from sepsis and exhaustion secondary to the long-standing disease.

CASE 3.—A man, aged 63 years, had had gallbladder symptoms intermittently for thirty years. The present attack had continued for ten days, having begun with a severe pain in the epigastrium, which radiated to the shoulder and was accompanied by gaseous distress and a rise of temperature. He had also been jaundiced and troubled with itching. In addition, he complained of some bladder retention.

A moderate tenderness over the upper abdomen was the only physical finding. The preoperative diagnosis was extrahepatic obstructive jaundice, probably caused by a stone in the common duct. Since the urinalysis revealed the presence of considerable pus, an in-dwelling catheter was used for two weeks, while a blood transfusion and other preoperative measures were carried out until the patient was in fit condition for exploration.

On opening the abdomen, the gallbladder was found to have perforated onto the pyloric end of the stomach, the perforation being protected by adhesions. Several stones were discovered in the abscess pocket, as well as in the gallbladder and common duct. A partial cholecystectomy was done, the common duct opened wide, the stones removed, and a T-tube inserted for drainage. Bile drained freely and the patient's convalescence was satisfactory for five days. Nausea, vomiting, and a decrease in the bile drainage then ensued for ten days, with a decline in his general condition. Intestinal obstruction was feared, but barium passed through the bowel, proving its patency. Following a blood transfusion, cellulitis with multiple abscesses appeared on his arm. On the twenty-sixth day after operation he developed bilateral bronchopneumonia and anuria, and died two days later.

CASE 4.—A man, aged 46 years, gave a history of intermittent attacks of epigastric pain, with an associated jaundice, during the previous five years. For ten months, his symptoms had been more severe, having indicated, at times, a peptic ulcer; at other times, they had been mixed, pointing to both ulcer and gallbladder disease. Coincidentally, he had gradually lost weight.

A moderate epigastric tenderness and a sense of thickening of the deep tissues were the only abnormal physical signs. The roentgenologist reported evidence of an obstructive lesion in the duodenum. The preoperative diagnosis was chronic cholecystitis and duodenal ulcer.

After two weeks of preparation, the abdomen was explored. The gallbladder was enlarged and subacutely inflamed and had perforated into the pyloric end of the stomach, as well as into the first and second portions of the duodenum, producing a natural cholecystogastrostomy and cholecystoduodenostomy. There was also a large gastric ulcer on the lesser curvature near the pylorus.

A cholecystectomy and partial gastrectomy were performed. The operation was extensive and prolonged, but in spite of this, the patient left the table in good condition, and his progress was satisfactory for twenty-four hours. He then became cyanotic, gasped, and died, presumably from pulmonary embolism.

CASE 5.—A man, aged 45 years, a stonecutter by occupation, had had a substernal abscess in childhood and in recent years several attacks of bronchopneumonia;

the last attack, seven months previously, had been complicated by pleurisy with effusion. During the prolonged convalescence, he had been seized with an acute epigastric pain accompanied by nausea and vomiting, which gradually subsided. A second and similar seizure had begun forty-eight hours before the patient's admission to the hospital.

On palpation, the patient's entire upper abdomen was tender, and the muscles were rigid. He had an elevation of temperature, and the leucocytes numbered 12,500. The urine contained both albumin and casts. The preoperative diagnosis was acute cholecystitis, pneumoconiosis, and acute nephritis.

Abdominal exploration revealed large old blood clots under the liver margin about the gallbladder. The latter organ was increased in size and filled with bloody fluid, and its wall was gangrenous. In its fundus was a perforation from which the blood had escaped into the peritoneal cavity.

A cholecystostomy was done and the abdomen freely drained. Two days later the lung condition became reactivated, the nephritis grew worse, and death occurred on the third day following operation.

CASE 6.—A man, aged 70 years, had suffered with symptoms of gallbladder disease for seven years. Five weeks before admission to the hospital, he had had a severe biliary colic with irregular fever, and had since lost weight and strength. He was also aware of dyspnea on exertion.

It was obvious that the patient was greatly debilitated. Weak heart sounds and an irregular pulse were significant of a damaged myocardium. The blood cell count showed 19,200 leucocytes. Marked tenderness and a suggestive mass were noted in the gallbladder region, and distinct muscle spasm with some rigidity spread over the abdomen. The preoperative diagnosis was acute cholecystitis with empyema and stones, and myocardial insufficiency.

Operation was carried out under local anesthesia. The gallbladder was distended with purulent fluid but contained no stones. Its walls were gangrenous almost throughout, secondary to vascular damage and congestion of the lymph spaces. A perforation was found in the fundus, through which infectious material had leaked into a pocket formed by the omentum.

A cholecystostomy was done and thorough drainage instituted. Soon after the operation, the patient developed an uncontrollable hiccough. He gradually declined and died on the sixth postoperative day. The cause of death was recorded as sepsis and myocardial insufficiency.

CASE 7.—A man, aged 59 years, had been seized with a violent, radiating epigastric pain, accompanied by nausea and vomiting, seven days before admission to the hospital. His condition was growing worse, and it was clear that a real intraabdominal catastrophe had occurred. This was his first attack; no history of previous gallbladder disease or biliary colic could be obtained.

The patient was acutely ill. Tenderness and muscular rigidity extended over the entire abdomen but were most pronounced over the gallbladder region and downward to the pelvis on the right. No mass was palpated. His temperature was slightly elevated, and the blood cell count revealed a moderate leucocytosis. The findings otherwise were not significant. The preoperative diagnosis was acute cholecystitis and acute appendicitis.

A high muscle-splitting incision was made rather far out to the right. A large amount of bile-stained fluid was seen to have gravitated along the outer side of the colon and into the pelvis. The appendix was not involved in his present complaint but was removed. A transverse incision was then made over the upper abdomen. The gallbladder was greatly distended and acutely inflamed; infected fluid was leaking from a small, unprotected perforation near the cystic duct. The blood

supply of the gallbladder was damaged but not destroyed. A local peritonitis involved the duodenum and contiguous tissue and apparently was spreading.

A cholecystectomy was done, ample drainage provided, and the abdomen was closed. Bile-stained fluid drained freely into the dressing but the patient grew increasingly restless; his temperature rose to 104.6 degrees; his pulse rate became rapid, irregular, and weak; and he died forty-eight hours after operation. An autopsy was not performed, but the causes of death were believed to be general peritonitis and acute myocardial insufficiency.

CASE 8.—A man, aged 61 years, had had several attacks of severe abdominal pain, which had begun nine years earlier and which had occurred at intervals for four years. During the following five years he had been free of discomfort until the present attack, two weeks before he entered the hospital. The onset had been sudden, with an excruciating pain in the epigastrium, which radiated to the shoulder. He had also had some fever and had been deeply jaundiced.

The abdomen was tender and tense; no mass could be palpated. The urinalysis indicated a mild infection of the urinary tract; otherwise, the examination was negative. The preoperative diagnosis was chronic cholecystitis.

At operation, the gallbladder was small and contained one large stone. It was adherent to the duodenum, into which it had perforated, forming a complete fistula. The common duct was opened and explored, but no stones were found. The head of the pancreas was nodular and inflamed and was probably responsible for the jaundice.

A cholecystectomy was done, the duodenum opening sutured, and the abdomen closed. The patient made good progress for a few days. He then began to grow weaker, and his urine output became increasingly less. All therapeutic measures failed to restore proper excretion, and he died twenty-two days postoperatively from uremia and exhaustion.

COMMENT

The proper time for operation in acute gallbladder disease has been much disputed. The main difficulty, in my opinion, is determining whether the condition is or is not acute. Even the keenest diagnostic acumen is fallible in discerning perforation in many instances. Those authors who have mentioned the matter have admitted that their preoperative diagnosis was correct in comparatively few cases of perforation of the gallbladder. As has been stated often, the symptoms are so variable in number and intensity that no certain syndrome can be regarded as pathognomonic of rupture until its devastating consequences become almost irreparable. Several cases of this series are proof that an acute perforation may be found when no history of an acute exacerbation of symptoms is given and the patient's condition is not extreme at examination. A scout film of the abdomen or fluoroscopic study may afford a clue to the diagnosis in revealing the presence of stones in the region of the gallbladder, but this is not entirely dependable. We know that perforation can and does take place when no stones are present. The four cases of gangrene with perforation mentioned in this series are examples.

Cases of chronic perforation are recognizable, as a rule, by a history of unchanging, more or less severe symptoms over an extended period. There is no need for delaying operation here. When acute symptoms have begun to subside, surgery should be postponed until further regression takes place. But if the patient's suffering is unduly prolonged or is increased, the condition may be considered acute, requiring immediate operation. Prompt surgery is indicated, of course, when persistent nausea and vomiting, weak pulse, increased respiration, an elevated leucocyte count, marked tenderness, and excruciating pain point to rupture into the peritoneal cavity.

In deciding the most opportune time for surgery, therefore, we can be guided only by present signs and symptoms. Operation may be elective in the apparently chronic cases, but it is imperative and urgent if symptoms already pronounced remain unchanged or become aggravated. Surgery may be postponed for a few days when there is evidence that the symptoms are subsiding or a possibility that they may do so under treatment. We agree with Behrend that, under proper control of a capable surgeon, there is little danger that an acute case of cholecystitis may go on to perforation; an exacerbation of symptoms may be readily detected and operation performed without delay.

It is generally conceded that cholecystectomy is the procedure of choice in gallbladder disease, when feasible. One material advantage of allowing acute symptoms time to subside lies in the fact that cholecystectomy usually may be done in the subacute stage, and thus the probable necessity for a second operation obviated.

In acute cholecystitis, when removal of the organ is contraindicated, Heyd and others have employed the method of bisecting the gallbladder from fundus to duct, enucleating the mucous membrane, inserting a tube, and closing the gallbladder about it. Heyd states that cholecystectomy is too dangerous and cholecystostomy alone often leads to the necessity for a second operation because of retention of an infected mucous membrane. Behrend believes the formation of adhesions the principal source of danger in a second operation. Both of these factors contribute to the hazard of cholecystectomy after cholecystostomy.

As was stated in the beginning, the whole purpose of this discussion is to dispel the illusion that perforation of the gallbladder is a negligible possibility and to bring to mind more forcibly the poor prognosis after its development. It is difficult to comprehend why, considering the dangerous potentialities, patients suffer with gallbladder symptoms for years before obtaining permanent relief. The fault cannot be attributed wholly to ignorance on the part of patients. Surely, many more are examined than are relieved. The responsibility lies

with us, then, who, realizing after a reasonable length of time that medical treatment is not curative, fail to see that our patients receive the benefit of surgery before the disease process progresses too far. An awakened conscientiousness on the part of medical men would seem to be largely effective in reducing the unnecessarily high number of perforated gallbladders and the appalling number of fatalities in their train.

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Editorials

Intestinal Obstruction: The Present Status of the Problem

FACTS are wrested from nature often, only after an arduous struggle. To this difficulty in the acquisition of wisdom, bowel obstruction has been no exception. No divining rod has been available to indicate where truths lay hidden. Critical and accurately made observations subjected to the crucial test of direct experiment are the only means by which new and basic principles are added to our knowledge.

For decades, clinicians and investigators have wrestled with the intangibles of bowel obstruction. Out of their diligent labors have emanated a few simple, but fundamental, truths. On reflection, some of these appear almost self-evident. Is not the same often said of most new discoveries? Today we hear less of the alleged toxic factor in acute intestinal obstruction and more concerning the effects of prolonged obstruction upon the bowel wall. The significance of the loss of water and electrolytes through the agency of vomiting is generally appreciated. Long-continued intestinal occlusion gives rise to increases of intraluminal tension which may threaten the viability of the bowel wall and increase its permeability. In this manner, a new route may be opened up for absorption, against which the body has developed no effective defense —transperitoneal migration of noxious agents. As long as the intestinal mucosa remains viable, this lining of the intestinal cylinder, it would appear, affords us the same protection in the presence of bowel occlusion which it bestows bountifully upon us all every day. With the onset of obstruction, bacteria multiply in the intestine, and the amicrobic character of the upper reaches of the upper gut becomes nonexistent. Belated recognition is also being accorded the blood loss factor which attends strangulating types of obstruction in which considerable blood may be lost into the infarcted segment of the bowel. In the avowal of these occurrences as consequences of obstruction, the therapeutic measures indicated are plainly suggested. (1) Adequate replacement of fluid loss by the liberal paraoral administration of saline solution in all instances in which vomiting has been a feature; (2) early relief of distention by measures which preserve the sterility of the peritoneal cavity; (3) restoration of blood loss by transfusion in strangulating obstructions. All these are matters of general agreement and need no amplification.

The manner in which relief of existing distention is to be attained is not so generally agreed upon. In the main, however, these differences

of opinion relate largely to the late case and the employment of compromise measures. To the early mechanical obstruction, by quite uniform consent, are applied the remedial measures of election. It is natural that amongst surgeons some controversy should hedge about the choice of procedure. It is equally fitting that as much thoughtful consideration be accorded the actual plan or design of an acceptable procedure. For undoubtedly the technique of execution of a procedure is fully as important and telling as is the choice of therapeutic expedient. Whether, in equally skilled and practiced hands, release of the bowel is secured by severing an obstructing band or performing enterostomy is often not an important issue. If, however, the bowel is punctured in freeing the gut or if the compromise measure of enterostomy is not *aseptically* done, the doom of the patient is sealed —not by the choice of remedial agent, but by the bungling of the surgeon. With release of the obstruction, no matter how great the distention, nor whether the obstructing agent be removed or only an exit be afforded for the removal of the retained gas and fluid, if the obstruction is simple in character and the bowel wall is intact and no visible soiling of the peritoneum has occurred, the outlook for the patient is promising. All strangulating obstructions operated upon early, with the single exception of thrombosis or embolism of the mesenteric vessels, may be treated as instances of simple obstruction after liberation of the constricting agent. In all instances in which the viability of the intestinal wall is lost, the threat to life is great. Operations for the relief of intestinal obstruction are accompanied by risk in so far as the sterility of the peritoneum has been vitiated by the condition of the bowel or violated by the surgeon. The operative procedure of least magnitude which will accomplish the desired end, when executed *aseptically* and with minimal trauma to the obstructed bowel, promises the patient the best chance for recovery.

The importance of early recognition of intestinal obstruction is acknowledged on every hand. There are obstructions, to be certain, which are notably difficult to recognize, such as an enteric intussusception or occlusion of the lumen of the bowel by a gallstone. Differentiation between an inflammatory intraperitoneal focus simultaneously interrupting the continuity of the bowel and a primary strangulating obstruction presents one of the most difficult problems in abdominal diagnosis and one which not uncommonly needs exploratory incision for its proper and safe solution. The larger number of bowel obstructions, however, can be accurately identified by a careful appraisal of the history, methodical employment of available criteria of physical examination, and the use of the scout film of the abdomen. When these findings are properly integrated, the examiner should be able to say with some confidence: (1) whether the bowel is obstructed; (2) whether the obstruction is simple or strangulating in character; and (3) whether it concerns the

large or the small bowel. He may often not be able to decide to his entire satisfaction just how the bowel is obstructed.

"Acute abdomen" is not synonymous with necessity for exploratory laparotomy, though, sad to say, it has too long been. The advent of constant recourse to x-ray examination (scout and erect films) in the acute abdominal lesion of perplexing origin has contributed much to correct diagnosis. The report of the roentgenologist, uninstructed as to the patient's history and physical findings, however, is often misleading and without value. When the roentgen findings are reviewed in the light of the clinical aspects of the case, however, they attain real significance. Visualization of gaseous distention in segments of the bowel attains significant meaning when coordinated with the presence or absence of vomiting, intestinal colic, abdominal tenderness, and gastric retention. No clinical diagnosis of bowel obstruction is complete if roentgen examination has been omitted.

In colonic obstructions, vomiting is often absent altogether; likewise, employment of the duodenal tube and suction demonstrate that gastric retention of fluid is almost invariably absent. In obstructions of the small bowel, on the contrary, copious and frequent vomiting is usual. Fecal-like vomiting is common in acute obstruction of the small bowel, but it has never been observed by the writer in colonic obstructions. Strangulating obstructions, in which blood escapes into the peritoneal cavity, invariably present rebound tenderness in addition to *intestinal colic* which accompanies all mechanical obstructions. The revelations of the scout film, when properly harmonized with the clinical findings, afford a stamp of finality to the diagnosis upon which reliance may be placed.

Within the past few years, a new agent has entered the lists as a rival, contending with operation for honors in the relief of intestinal distention due to obstruction—suction applied to an indwelling duodenal tube. Those who have schooled themselves in the intricacies of the art of recognition of acute abdominal disease have found the method helpful and a satisfactory substitute for certain obstructions for which they were previously wont to perform operation. In adhesive, incomplete occlusions and in physiologic obstructions, the method has found its greatest usefulness. As an auxiliary to surgery, it has proved of value in most obstructions. Its limitations are quite understandable and the employment of suction applied to an inlying duodenal tube as the sole remedial agent is strictly contraindicated essentially in two kinds of obstruction: (1) strangulating obstructions, (2) occlusion of the left colon in which the competent proximal ileocolic sphincter limits the distention to the colon. He who essays to use the plan as a trial method in diverse conditions to determine whether operation will be necessary is likely to encounter difficulties. Moreover, mere cessation of *intestinal*

colic does not suffice to indicate that a satisfactory decompression is being achieved. Dependable information is to be obtained only by subsequent check-up scout films made at twelve- to twenty-four-hour intervals.

That progress has been made in the management of intestinal obstruction is attested in the recitals which appear in this issue of SURGERY from the pens of several men who have striven long with the problem. Wisdom is compounded of many experiences. Much yet remains to be learned. From the successes of these labors let us take courage and gird ourselves for a renewal of the conflict with the obstruction problem.

—*Owen H. Wangensteen.*

Transfusion Syphilis

IT IS well known that the virus of syphilis may be transmitted by blood transfusion. This *syphilis d'emblée* becomes apparent in from one to three or four months after the transfusion with the development in the recipient of the acute skin and mucous membrane lesions of the secondary stage of the infection. Although the literature contains relatively few case reports of such therapeutic accidents, they occur frequently enough to have become a part of the experience of most active hospital groups. The surgeon especially is likely to be *particeps criminis* since he commonly employs blood transfusions under circumstances of stress and emergency, when the detection of syphilis in the donor through careful questioning and physical and serologic examinations is difficult or impossible.

It is clear that the virus of syphilis must be present in the blood of the donor if transfusion is to result in syphilitic infection in the recipient. In experimental syphilis, it is known that the virus is present in the blood of rabbits during the acute or active phase of the infection and that with the disappearance of active lesions the blood loses its capacity to infect.[1] This would seem to be the case in the human disease. Eberson and Engman[2] injected blood from seventy-three individuals with chronic syphilis into rabbits' testicles and not a single infection resulted, although with similar technic blood from patients with acute syphilis regularly induces infection. There are several reported instances of the use of blood from donors with chronic syphilis for transfusions in which the disease was not transmitted to the recipients. A review of the reported instances of "transfusion syphilis" reveals that with one exception[3] all of the donors either had acute syphilis or were pregnant syphilitic women. In this case it seems probable that the donor was experiencing relapsing, acute syphilis at the time he gave

blood for transfusion. The use of chronic syphilitics as donors, however, is a procedure not to be recommended except under most urgent circumstances and then only when blood from other sources is not available.

The serologic tests are not infallible indicators as to the presence or absence of *Treponema pallidum* in the donor's blood. The reactions may be positive when organisms are not present in the blood, as in chronic syphilis, and may be negative when the blood is teeming with *Treponema pallidum*, as in the incubation and seronegative stages of acute syphilis. The absence of recent exposure to syphilis and of chancre of skin or mucous membrane constitute our only guarantee against the existence of the latter circumstance in a prospective donor.

To avoid "transfusion syphilis" in emergency treatment one must, therefore, question the prospective donors relative to recent exposure, thus establishing facts relative to the possible existence of the stage of incubation, and one must examine for chancre (dark-field examination of any suspicious lesions), adenopathy, rash and mucous membrane lesions. If the history and these simple and readily made examinations are negative and the donor is not a pregnant woman, the very great probability is that syphilis, if present, is not in the acute stage and therefore not transmissible by blood transfusion. A careful, accurate history and examination of the body surfaces and orifices of the prospective blood donor are, therefore, more important than the serologic tests with donors' serum, valuable as these are. When reliance is placed solely on the Wassermann or Kahn test, false security may be encouraged even though the disease is present and transmissible to the recipient, as in the reported instances of "transfusion syphilis" induced by the use of donors in the incubation stage of the disease.[4]

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Recent Advances in Surgery

CONDUCTED BY ALFRED BLALOCK, M.D.

THE USE OF PARENTERAL FLUIDS

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(From the Joseph Hume Research Laboratory of Touro Infirmary)

WATER is of utmost importance to the living organism. It forms part of every living cell and provides the medium in which the chemical reactions of the body occur. It constitutes more than one-half of the body by weight. Sir Arthur Shipley states that "even the Archbishop of Canterbury comprises 59 per cent of water."[1]

Clinicians have long recognized that not infrequently the life of the patient depends upon the maintenance of an adequate water balance. New impetus to interest in problems of hydration has been furnished by the accurate and very practical studies of Newburgh, Coller, Maddock, and their coworkers.[2-8] There are now available methods of calculating the fluid requirements of patients in all stages of fluid deprivation. It is the purpose of this paper to discuss the problem of hydration of patients with special reference to the physiologic considerations which determine the amounts and kinds of solutions to be administered.

Fluids are available to the body in the form of fluids ingested as such (about 1,500 c.c. per day), in the water content of solid food, and from water of oxidation. So-called solid foods are made up largely of water; e.g., a pound of steak will yield about 325 c.c. of water; the average maintenance diet supplies about 1,000 c.c. of water daily. Oxidation of the organic matter or of body tissues in starvation produces about 500 c.c. more.[9-11]

In disease states it may not be possible or permissible to supply fluids by the gastrointestinal tract, and adequate fluid and salt intake becomes the physician's problem. Intelligent management of this problem depends upon an understanding of the nature and quantity of the fluid losses and the proper interpretation of the disturbed physiology produced by their loss.

In the normal individual water is lost from the body by three routes, viz., the kidney, the skin (and lungs), and the gastrointestinal tract. The burden of the excretion of several grams of waste products is thrown upon the kidney. In order that these waste products be entirely elimi-

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nated, a certain minimal amount of water is required to carry them out. The healthy kidney can concentrate urine to a specific gravity of approximately 1.032. To carry out the average daily waste products (35 gm.) in a maximally concentrated urine, about 500 c.c. of water are required. When renal function is so impaired that the kidney has little or no concentrating power, about 1,500 c.c. of water are required.[11, 12] If the water available for urine formation is inadequate to care for the removal of the waste products, these accumulate in the body fluids.

In the allotment of available fluid, the kidney is favored to a lesser degree than the other body tissues. The skin and lungs (water for vaporization), the gastrointestinal tract (diarrheas, vomiting, etc.) and the interstitial tissues (during edema formation) enjoy preferential rights over such fluid as is available, and their demands are satisfied first. The kidney uses the fluid which remains. The diversion of fluid for purposes other than urine formation has been called "prerenal deviation of fluids."[13]

These considerations have certain important implications. The most clinching evidence that the normal organism is not receiving enough water is the finding of a twenty-four-hour urine output of less than 500 c.c. If renal function is good, the urine will be highly concentrated (specific gravity 1.020 or higher). A small twenty-four-hour urine output with high specific gravity is (in the absence of edema formation) sufficient evidence for making a diagnosis of dehydration. As dehydration continues, beginning nitrogen retention may be noted. In extreme cases urinary secretion may cease completely. The skin is hot and dry; the urine is scanty and contains protein, red blood cells, and casts in greater than normal amounts; marked nitrogen retention augmented by urea from the increased protein catabolism in dehydration[14] is usually present. Too frequently this syndrome is unjustly labeled toxic nephritis. The whole picture can be produced in normal persons by simply depriving them of fluid[11] and the remedy is, of course, administration of adequate quantities of fluid.

When the concentrating power of the kidney is reduced, the specific gravity of the urine of severely dehydrated patients will be lower than 1.020 (with maximal impairment, as low as 1.010) and the problem of determining whether the nitrogen retention is caused by dehydration or primary renal insufficiency may require further study. An approximate calculation of the fluid balance of the patient for a few preceding days may yield valuable information.

As will be indicated in the following sections, losses of water and salts, in excess of body economy, occur by way of the skin and gastrointestinal tract. Only rarely is the kidney the primary route for depletion of salt and fluid. Two well-known instances are diabetes mellitus and terminal nephritis. In the former, excessive urinary output of sugar and acidosis cause a profound diuresis with loss of body water and salts; in the latter,

the damaged kidney is unable to economically regulate salt and fluid output. Since the incidence of primary renal depletion of salt and water is relatively small and requires consideration beyond the scope of this paper, these obligatory polyurias will not be discussed further.

The Skin (and Lungs) as a Route of Depletion of Body Water and Electrolytes.—Heat is lost from the skin and lungs by convexion, conduction, radiation, and by evaporation of water. Heat lost by the evaporation of water is associated with loss of body fluid, the heat being dissipated in changing water from the liquid to the gaseous state. The water which is so vaporized, in the absence of visible perspiration, is called insensible water.[16] The heat used in vaporizing this water amounts to about 24 per cent of the total heat eliminated from the body.[8] Richardson[17] has shown that this water loss is independent of the sweat glands, for about 28 per cent of the total heat was lost by vaporization of insensible water in a patient with congenital absence of the sweat glands. Thus, maintenance of a constant body temperature obliges an animal to lose a certain amount of water so that heat may be dissipated. This obligatory water loss in the normal individual amounts to 1,000 to 1,500 c.c. per day.[2] This water loss is associated with a negligible loss of electrolytes, for Benedict[19] found that only 1.15 gm. of chloride were lost from the skin during a fasting period of 27 days.

When there is increased need for eliminating heat, the sweat glands function actively, elaborating fluid on the surface of the body. Evaporation of this fluid from the skin is effective in eliminating heat except when the sweat is lost from the body surface without being evaporated. Whereas, insensible water loss is of importance in calculating the fluid balance of the patient, it is never the primary cause of severe dehydration and depletion of body electrolytes. Such is not the case with the loss of fluids and electrolytes by the sweat.

Prolonged, profuse sweating can occasion the loss of tremendous quantities of fluid and electrolytes producing marked depletion of body salt and water.[15, 20, 21] While the skin may exert some slight ability to spare body salt,[15] its efficiency in this capacity is negligible. In profuse sweating as much as 2 gm. of sodium chloride may be lost from the skin per hour. The salt is excreted in a hypotonic solution (about 0.2 per cent sodium chloride[20]). Since the sweat is derived from the serum, each liter of sweat depletes the body of a liter of water. Because the salt concentration of the secreted water is considerably less than that of the body fluids from which it was derived, there is more water lost than salt. The excessive amount of salt cannot be retained by the tissues without water to render it isotonic, so it is eliminated by the kidney with minimal loss of water.[22] Ultimately then, a liter of sweat deprives the body fluids of a liter of isotonic saline. The electrolyte pattern of sweat resembles that of serum so that the loss of sweat is not associated with disturbances of the serum electrolyte pattern.

The dehydrating effects of profuse sweating may be alleviated by oral administration of 0.2 per cent salt; if water is ingested without salt, the reduction of the osmotic pressure of the body fluids causes the onset of the syndrome known as miner's cramps.[20, 21] If for some reason fluids cannot be taken by mouth, parenterally administered isotonic saline serves the same function admirably.

Glucose solutions cannot serve to replace the body losses due to profuse sweating because they do not supply the needed electrolytes and the water is not retained without salt to make it isotonic. Glucose solutions do, however, serve admirably as a source of water for vaporization needs which, in certain pathologic states, may be as great as 2,500 c.c. per day.[2, 6]

The Gastrointestinal Tract as a Route of Depletion of Body Water and Electrolytes.—The fate of the digestive secretions is of prime importance in any consideration of body dehydration. The magnitude of the fluid and electrolyte mobilization is often underestimated and abnormal deviation of these fluids and electrolytes leads to clinical syndromes requiring more or less specific therapy necessarily based on the disturbed physiology.

The gastrointestinal tract consists of a hollow tube, the lumen of which is outside the body tissues. Into this canal are poured daily seven to ten liters of digestive juices. Secretion of these juices occurs almost entirely into the proximal portion of the canal. The juices (saliva, gastric juice, bile, pancreatic juice, and succus entericus) consist of water, electrolytes, and enzymes. They serve their functions in the canal and are propelled by the intestinal movements to the distal portions of the canal for reabsorption. The secretion and absorption of the digestive juices constitutes an internal circulation of great efficiency. By the time the intestinal contents have reached the sigmoid colon, the inspissated fecal material contains only a few cubic centimeters of water and small amounts of salts. The daily loss of water in the stool is about 100 c.c.

Two features of the internal circulation are significant from the standpoint of economy of body fluids and electrolytes:

1. The origin and chemical composition of the digestive secretions: The digestive fluids are derived from the blood serum and hence from the interstitial fluids with which they are approximately isotonic. The secretion of a liter of digestive secretion into the intestinal canal—lying outside the body tissues—is equivalent to depleting the body fluids of a liter of isotonic fluid. While the digestive secretions are isotonic with the serum electrolytes, they vary considerably in chemical composition; e.g., the gastric juice is richer in chloride and poorer in base than is serum. Secretion of gastric juice dehydrates the body and leaves the serum richer in base and poorer in chloride. Pancreatic juice con-

tains less chloride and more bicarbonate than serum, and secretion of pancreatic juice dehydrates the body and leaves the serum relatively richer in chloride and poorer in bicarbonate (Tables I and II).

2. The mechanics of the internal circulation make it vulnerable to pathologic variations associated with dehydration and electrolyte depletion:

a. Removal of secretions from the proximal portion of the gastrointestinal tract as occurs in vomiting, gastric and duodenal fistulas, high intestinal obstruction.

b. Failure of transport of the secretions to the absorptive area as occurs in ileus, acute gastric dilatation, intestinal obstructions.

c. Short-circuiting of the secretions to the outside as occurs in biliary, pancreatic, and intestinal fistulas.

d. Abnormal hyperactivity of the bowel as occurs in the diarrheas.

TABLE I

CHANGES IN GASTRIC JUICE AND SERUM ELECTROLYTES DURING DIGESTION AND PYLORIC OBSTRUCTION*

	Total Base	Cl	HCO_3
	M-Eq	M-Eq	M-Eq
Gastric juice—fasting	95	155	–
Gastric juice during secretion	38	150	–
Serum electrolytes—normal dog	160	112	28
Serum electrolytes of same animal 66 hours after pyloric obstruction	138	40	38

*Modified from data of Gamble and McIver[30] and Gamble and Ross.[25]

Because of differences in chemical composition, the loss of the digestive secretions proximal to the pylorus will produce body disturbances differing from those occasioned by the loss of digestive secretions secreted distal to the pylorus. The following considerations, bearing upon the continued loss of gastric juice, are of importance in considering the pathologic physiology of pyloric and high intestinal obstruction, gastric and duodenal fistulas, acute gastric dilatation, and vomiting.

The intimate relationship of the secretion of the gastric juice to the serum electrolytes is evidenced even during normal digestion. At such times, the gastric juice contains less base than fasting gastric juice (Table I). The variation in acidity is caused by the retention of base within the blood during secretion of the juice so that blood leaving the stomach is richer in base than blood coming to the stomach.[23] The extra base is excreted in the urine and accounts for the alkaline tide which is not observed in individuals with achlorhydria.[24] Under normal circumstances the acid gastric juice is neutralized distal to the pylorus and the fluid and electrolytes are reabsorbed to restore tissue losses. If now the transportation of the secreted gastric juice to the absorptive area is arrested or if the juice is ejected from the proximal portion of

the canal—as occurs in the above mentioned pathologic states—the continued loss of gastric juice renders the serum poorer in chloride and richer in base and the tissue fluids are depleted of fluid and electrolytes. The production of this picture in pyloric obstruction may be seen in Table I. Total base is depleted; the chlorides drop to less than half their normal value; and the retained sodium combines with the carbonic acid formed during metabolic processes in tissues to increase the level of bicarbonate. Increases of bicarbonate to tetany levels (45 mm.) have been observed. In addition, vomiting is common and nutrition cannot be maintained so that carbohydrate deprivation and starvation ketosis may occur. The prerenal deviation of fluid may be of such magnitude as to leave too little water for adequate urinary volume and nitrogen retention may be present. The clinical picture includes: (1) dehydration, (2) depletion of body electrolytes, (3) imbalance of serum electrolytes (alkalosis), (4) starvation ketosis, and (5) azotemia.

Cases with achlorhydria will show the same picture with the exception that they do not develop alkalosis because sodium and chloride are lost in equivalent amounts.[29]

Fluid therapy should include parenteral administration of saline solutions (to restore fluid, total electrolytes and to restore the electrolyte pattern) and glucose solutions (to relieve ketosis and excessive protein catabolism, supply ample fluids for urine formation to remove the accumulated nitrogenous wastes). Each type of fluid serves a specific function and both are needed to restore equilibrium. With an adequate supply of fluid the kidney quickly removes the nitrogenous wastes and corrects the electrolyte pattern by excreting sodium bicarbonate in the urine and retaining sodium chloride which the body needs.[25, 26]

Similar serum electrolyte patterns and dehydration occur when the secreted juices are not vomited but are retained in the stomach and proximal portion of the gastrointestinal tract.[27, 28] Their presence in the upper part of the tract is associated with marked nausea. Absorption of the secretions relieves the nausea and restores the serum electrolytes to normal.[28]

TABLE II

A COMPARISON OF CERTAIN ELECTROLYTES IN BLOOD SERUM AND IN THE DIGESTIVE SECRETIONS*

	Total Base	Cl	HCO_3
	M-Eq	M-Eq	M-Eq
Serum	164	110	22
Fundus gastric juice	40	160	–
Pylorus gastric juice	188	142	–
Duodenum	177	120	–
Jejunum	155	150	20
Ileum	162	80	90
Colon	160	80	92
Pancreatic juice	168	115	45
Hepatic bile	182	90	45

*Modified from data in Peters' *Body Water.*[49]

Gamble and his coworkers[30-32] have shown that the total electrolyte concentration per unit of water in all the gastrointestinal secretions very closely approaches that in the blood serum. In Table II it may be seen that while the concentration of total base in the intestinal secretions below the pylorus very closely approaches that in serum, the pancreatic juice, hepatic bile, ileum and colon secretions contain increasing quantities of base combined with bicarbonate and decreasing amounts combined with chloride.[33] In general, the loss of the digestive juices of the small intestine and colon will entail the loss to the body of isotonic fluid containing total base and acid radicals made up predominantly of bicarbonate and to a lesser extent of chloride. Consequently imbalance of serum electrolytes occurs in which bicarbonate is depleted and chlorides may be normal or increased in concentration.

The above facts are of significance in interpreting the effects of the loss of these secretions in severe diarrheas and low intestinal fistulas. It has long been known that severe diarrheas (cholera, bacillary dysentery, amebic dysentery, diarrheas of infancy) may be the cause of serious dehydration. Fluids and electrolytes are hurried past the absorptive area so rapidly that absorption is at a minimum. The output of total base may greatly exceed the intake.[34] In low intestinal fistulas where fluid is short-circuited to the outside, the events are less acute but similar in direction.

While there is some controversy as to the incidence of hyperchloremia,[35-38] it is agreed that dehydration, electrolyte depletion, and depletion of bicarbonate do occur. In addition, starvation ketosis and nitrogen retention may be present.

Saline solutions should be administered parenterally to replace body fluid and electrolytes and glucose solutions are needed for relieving ketosis and dehydration. Hartmann[37] believes that bicarbonate should also be given, for he found that saline and glucose alone do not restore bicarbonate to normal and may even exaggerate the chloride excess. Hoag and Marples,[35, 36] on the other hand, find that if enough fluid is given to maintain adequate urinary output, there exists neither a need nor an indication for administering bicarbonate. Peters and van Slyke[39] sum up the matter by stating that "although bicarbonate may be of value in expediting the restoration of the normal electrolyte pattern by filling a specific deficiency, it seems to be less important than the establishment of normal kidney function by adequate supplies of water and salt. Bicarbonate can be produced in the body . . . . chloride deficiency can only be met by administration of chloride." They suggest the following as a possible mechanism whereby the body restores depleted bicarbonate from administered saline:

$$\underset{\text{formed in kidney}}{(NH_4)HCO_3} + NaCl \longrightarrow \underset{\text{excreted in urine}}{NH_4Cl} + \underset{\text{retained in body}}{NaHCO_3}$$

In obstructions of the small intestine and in ileus, vomiting is usually a conspicuous feature and the loss of acid gastric juice may not only balance the bicarbonate losses of the other juices but may surpass them to produce the blood electrolyte pattern found in high intestine obstruction. The site of the obstruction, the extent to which absorption is impaired, and the nutritive state of the patient are uncontrolled factors which make prediction of the electrolyte pattern hazardous. If, however, the obstruction is distal to the absorptive area (at the level of the sigmoid colon), there may be little interference with the absorption of the digestive secretions and orally administered fluids.[40]

Thus, disturbances of the gastrointestinal tract may lead to serious depletion of body fluid and electrolytes. When glucose solutions are administered, they provide water for vaporization and adequate urine output and carbohydrate to relieve ketosis and spare body protein; but when the sugar is utilized the water cannot be retained to replenish the depleted body stores because there is no salt to render it isotonic. From the standpoint of the body fluids and electrolytes, glucose solutions are useless for they neither replenish the body fluid nor the body electrolytes. For this purpose, saline solutions are indispensable. When the organism is well supplied with salt and water, the kidney exhibits a remarkable capacity to correct the electrolyte pattern. Excessive bicarbonate and diminished chlorides are cared for by retention of NaCl and excretion of $NaHCO_3$ in the urine; when base and bicarbonate are diminished and chlorides excessive, the $NaHCO_3$ is retained and the NaCl is excreted in the urine. The administration of additional bicarbonate in cases of chloride acidosis may be of some value.

While the normal obligatory water loss from the intestine is so small as to be negligible, in pathologic conditions associated with disturbances in fluid absorption, it may amount to several liters a day and constitute an important route of water and salt loss.

The Quantity and Quality of Fluids to Be Administered.—The quantity of fluid required by a normal individual is that amount which will supply the needs of vaporization and urine formation. It makes no difference to the body whether this amount of fluid is taken by mouth or is slowly administered parenterally in isotonic solution. The amount required per day is about 3,500 c.c. (vide infra). We are indebted to Coller and Maddock for a splendid solution of the problem of hydration requirements of patients with disorders of fluid balance. They found that the requirement of water for vaporization (for the regulation of body temperature) is about 1,000 to 1,500 c.c. in most patients, but under certain conditions (profuse sweating, hyperthyroidism) the amount needed may be as much as 2,500 c.c.[2, 6] On the average, 2,000 c.c. is adequate for the vaporization needs of almost all patients.

The amount needed for urine formation (1,500 c.c.) was calculated from data supplied by Lashmet and Newburgh.[12] The latter found that with minimal concentrating power the kidney requires about 1,500 c.c. of water to eliminate the average amount of waste products (about 35 gm.). Although the normal kidney can eliminate this much waste in about one-third this volume of water, an allowance of 1,500 c.c. will cover all ranges of renal function. Thus 3,500 c.c. of water are needed per day to ensure hydration in patients who are not dehydrated and have no abnormal losses (vomiting, diarrhea, fistulas, etc.).

The quality of the fluid to be administered parenterally to supply the water needs of the body has been the subject of considerable study. Unsuccessful fluid therapy prevails when patients are not analyzed with reference to their hydration needs and there is a failure to appreciate the disturbed physiology. Two types of isotonic solutions are in common use:

1. Sugar solutions	5 per cent glucose in water
2. Salt solutions	a. 0.9 per cent sodium chloride
	b. Ringer's solution

Both types have their indications and limitations determined by the existing needs of the patient. The needs of patients are readily recognized if patients are classified as follows:

Group 1.—This group includes those patients who require water only to meet the requirements of water for vaporization and water for urine formation. Coller, Dick, and Maddock[18] studied the effects of administration of both types of solutions to such a group "who had not lost appreciable amounts of sodium chloride but, because of their disease or because of treatment, could not or were not permitted to take sufficient fluids by mouth to maintain a normal water exchange." None of the patients were dehydrated; there were no abnormal losses; and most of them had little or no disturbance of the blood electrolyte pattern. The amount of each solution given was about 3,500 c.c. per day—2,000 c.c. for vaporization and 1,500 c.c. for urine formation. When 5 per cent glucose alone was given, all the patients lost weight because the caloric intake was below the requirement. When salt solutions were given for several days, the healthy patients did not retain salt and water but the sick patients did show evidence of water retention (by increase in body weight) even to the point of frank edema. Since these patients were not dehydrated, the retained water was not needed by the organism.

These results invite further consideration. When glucose solutions are administered, the carbohydrate is metabolized and the water is necessarily excreted because it does not contain salt to render it isotonic. Because the carbohydrate is metabolized, the administration of glucose solutions really means the administration of water to the organism. Strauss[41] has shown that water administered to normal individuals is

rapidly and completely eliminated and that its elimination is associated with the loss of only a small amount of electrolytes. Thus, glucose solutions make ideal hydrating solutions, for they are not retained and they wash out negligible quantities of body salt.

An equal amount of isotonic salt solution given to normal individuals is only gradually eliminated "presumably because it causes minimal alteration of the composition of the serum."[49] Baird and Haldane[45] found that even healthy normal individuals will develop edema after the continued daily administration of 35 to 40 gm. of sodium chloride per day (an amount contained in about 3,500 c.c. isotonic saline). This finding alone should be sufficient to indict the common hospital practice of administering several liters of isotonic saline for several days to patients who have not lost salt but who merely require water to meet their hydration needs.

The more rapid retention of salt and water by sick patients constitutes a difference from healthy patients of degree rather than kind. Somewhat similar retention has been noted in pneumonia,[42] tuberculosis,[43] and other conditions.[44] While impaired cardiac and renal function and low plasma proteins predispose to fluid and salt retention, these factors apparently played no rôle in the series observed by Coller, Dick, and Maddock.[18] The incidence and dangers of edema of the parenchymatous and subcutaneous tissues following prolonged intravenous saline therapy have been noted by others.[46, 47]

In view of the fact that electrolytes are not needed to satisfy the hydration requirements of this group of patients and may even produce edema in some, 3,500 c.c. of 5 per cent glucose may be used to advantage, for it supplies about 175 gm. carbohydrate to combat ketosis and spare body protein, and the water is ample for the needs of the skin and kidney. The continued administration of 5 per cent glucose is not associated with appreciable electrolyte loss, for Gamble, Ross, and Tisdall[48] have shown that in starvation, but without water deprivation, about 0.2 gm. sodium chloride is lost in the urine per day.

Group 2.—In this group are included those patients who are not dehydrated but who require parenteral fluids not only for normal hydration requirements but also to keep pace with abnormal fluid losses. Abnormal fluid losses include losses from the skin (excessive sweating), gastrointestinal tract (fistulas, vomiting, diarrhea, etc.) and the kidney (obligatory polyurias). The renal losses will not be discussed in this paper.

The skin losses due to excessive sweating require that some salt be replaced. Sweat is a hypotonic solution[20] containing about 0.2 per cent sodium chloride. As has been noted above, the salt left behind during sweating is excreted by the kidneys so that the end-result is the loss of a liter of isotonic saline for every liter of sweat. The losses from the gastrointestinal tract occasion the loss of fluids approximately iso-

tonic with the blood serum. They should be accurately measured and replaced liter for liter by the parenteral administration of isotonic saline. Glucose solutions are useless for these purposes, for while they supply water for vaporization and urine formation, they do not relieve the underlying defect—a need for electrolytes and retained fluid to restore the body water and electrolytes and the electrolyte pattern. The mechanism by which this is accomplished has been discussed in a preceding section of this paper. Patients in Group 2 will require not only 3,500 c.c. 5 per cent glucose to maintain hydration but also should have their losses due to prerenal deviation of fluids made good by an equivalent amount of saline.

Group 3.—This group includes all patients who are dehydrated. In an investigation of the amount of fluid which must be lost from the normal body to produce the syndrome of dehydration, Maddock and Coller[11] found that dehydration symptoms, including scanty urine of high concentration and beginning nitrogen retention, appear when approximately 6 per cent of the body weight has been lost. On this basis, they calculate that a dehydrated patient weighing sixty kilograms requires 3,600 c.c. of fluid to rehydrate his body tissues. Fluid deprivation to the point of dehydration from any cause is always associated with salt loss. The quota necessary to rehydrate the patient should be supplied by isotonic saline. Thus, in the first twenty-four hours, the above patient will require 3,600 c.c. isotonic saline for rehydration and 3,500 c.c. 5 per cent glucose to maintain hydration (as in Group 1); if there are abnormal losses, additional saline may be required (as in Group 2).

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Review of Recent Meetings

REPORT OF THE MEETING OF THE AMERICAN ASSOCIATION FOR CANCER RESEARCH, CHICAGO, ILL., MARCH 24, 1937

E. T. Bell, M.D., Minneapolis, Minn.

(From the Department of Pathology, the University of Minnesota)

1, **Dr. Wm. H. Woglom.** In his presidential address, Dr. Woglom discussed the mechanism of immunity to a transplanted sarcoma in rats. In rats which recover from a transplanted tumor, the growth increases until about the tenth day, after which it gradually recedes. In such animals, he obtained an immune body from the spleen which inhibited the growth of the tumor when incubated with it for some time. He demonstrated that a water-soluble, immune body was found in the spleen.

2, **Dr. E. T. Bell** and **Dr. T. P. Rothnem.** Two brothers, aged sixteen and thirteen years, developed extensive squamous cell carcinoma of the lower lip on the basis of xeroderma pigmentosum. The skin of the face, neck, and hands showed a mild degree of the disease. The tumors of the lip were apparently completely cured by x-ray treatment.

3, **Dr. Edgar H. Norris.** A large benign thymoma was found in a case of myasthenia gravis. The thymoma was interpreted as a localized hyperplasia of the thymus.

4, **Dr. Arthur H. Wells.** A classic case of pheochromocytoma of the adrenal was reported. The classic picture of paroxysmal hypertension was present. The tumor was found at postmortem. The literature contains nine reports in which paroxysmal hypertension was cured by removal of a benign pheochromocytoma of the adrenal.

5, **Dr. John J. Bittner.** Dr. Bittner reported a highly interesting study on the spontaneous carcinoma of the breast in mice. It had already been shown that mating of a female from a high cancer strain with a male of a low cancer strain gave a high incidence of carcinoma of the breast in the offspring, while mating of a high cancer strain male with a low cancer strain female gave a low incidence of carcinoma of the breast. The incidence of carcinoma of the breast in the adult breeding females of the high cancer strain was about 83 per cent. When the young of the high cancer strain are transferred immediately after birth and are nursed by a foster mother of a low cancer strain, the incidence of spontaneous carcinoma of the breast in these mice was markedly reduced. This low cancer incidence had persisted for seven generations. The author suggested that the stimulus to cancer was transmitted by the milk.

6, **Dr. Albert E. Casey.** Dr. Casey studied various malignant diseases of the lymphatic system and concluded that the length of life was inversely proportional to the percentage of mitoses found in microscopic sections. He estimated the percentage of mitoses by counting 1,000 nuclei.

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7, **Dr. Austin M. Brues** and **Dr. Elizabeth B. Jackson.** It was shown that colchicine applied to growing tissues increases the number of visible mitotic figures by preventing the formation of the spindle. The chromosomes become scattered through the cell and cell division does not occur. This causes an increase of the number of cells seen in mitoses at any one time.

8, **Dr. William S. Murray.** Dr. Murray studied the effects of parabiosis in mice of a strain which had a very high incidence of spontaneous carcinoma of the breast. Parabiotic pairs were made by anastomosing male and female mice at the age of one month. There was good intermingling of the circulation. There was a very high mortality up until a period of three months, after which they became adjusted, and a number were observed for one year or more. He noted that the females go through an almost constant estrus. There is a very marked hypertrophy of the ovarian follicles, but after a few months the follicles begin to degenerate. The testes were inhibited only slightly in their development. No corpora lutea were found. The female breast did not develop. No cases of cancer of the breast occurred. Apparently the male hormone suppresses the action of the female hormone.

9, **Dr. C. C. Little.** Dr. Little discussed the genesis of structural abnormalities in a black strain of mice. He noted that some abnormalities follow a simple mendelian inheritance and that others follow a mendelian inheritance with some variation. The third group is inherited irregularly, and in the fourth group there is little or no genetic influence and the abnormality is interpreted as a somatic variation. The cancers, in general, follow mendelian inheritance with some variations. A large number of abnormalities appear sporadically in a supposedly genetically homogeneous strain. He believes that these rare abnormalities are not determined by genes.

10, **Dr. Maud Slye.** Dr. Slye gave the results of many years of study on the inheritance of cancer in mice. She believes that the development of cancer is determined by a unit recessive factor. One factor determines the type of cancer and the other factor determines its location. In a very large amount of material she found that the actual incidence of cancer corresponded rather closely to the predicted incidence.

AMERICAN ASSOCIATION OF PATHOLOGISTS AND BACTERIOLOGISTS, CHICAGO, ILL., MARCH 25 TO 26, 1937

E. T. Bell, M.D., Minneapolis, Minn.

(From the Department of Pathology, the University of Minnesota)

1, **Dr. John Howe.** Dr. Howe found marked variations in the reaction to tuberculin from time to time in the same individual. He thought that the more intense reactions occur with the lower levels of diastolic blood pressure.

2, **Dr. Max B. Lurie.** Dr. Lurie found that in experimental inflammation in a tuberculous animal the exudate consists largely of mononuclear cells, even though the reaction of the exudate was alkaline. Menkin had noted previously that in the early phases of inflammation the reaction is alkaline and the exudate consists largely of polymorphonuclear leucocytes, but later the reaction became acid and the polymorphonuclears were replaced by mononuclears. Lurie found that this did not occur in the tuberculous animals.

3, **Dr. C. E. Woodruff** and **Dr. H. S. Willis.** Guinea pigs were inoculated with virulent tubercle bacilli, after which they became allergic. One group was desensitized with tuberculin. They found that the desensitized animals did not live as long as the allergic controls.

4, **Dr. Albert B. Sabin** and **Dr. Peter K. Olitsky.** The authors studied the route of extension of stomatitis virus and the virus of equine encephalomyelitis. Mice were inoculated in various parts of the body and the route of extension to the central nervous system was determined. When inoculated intranasally, the virus goes through the olfactory nerves to the brain and follows the olfactory pathways in the cortex. After intraocular inoculation, it travels to the opposite colliculus. The route of the virus in the brain is so definite that it affords a means of identifying intracranial nerve tracts. After intramuscular inoculation, the stomatitis virus goes by the peripheral nerves to the central nervous system. The virus of equine encephalomyelitis passes from any intramuscular site by the blood stream to the mucosa of the nose and thence by the olfactory paths to the brain. Application of tannic acid to the nasal mucosa prevents the development of equine encephalomyelitis after an intramuscular inoculation. Apparently the viruses spread along the axones of the nerve fibers.

5, **Dr. Cornelius S. Hagerty.** Dr. Hagerty produced embolic glomerular lesions by intravenous injections of fats and salts. In one type of lesion there was an endothelial proliferation and in another there was formation of collagen in the glomerulus. The author thought that the collagen was derived from fibrocytes which were present in the glomerulus.

6, **Dr. Benjamin Castleman** and **Dr. Tracy B. Mallory.** The authors reported ten new examples of adenoma of the parathyroid glands and two cases of diffuse hyperplasia. In the case of adenoma, a single gland is involved. These may be cured by removing the adenoma. Diffuse hyperplasias involve all of the parathyroid bodies and are secondary to some metabolic disturbances. Removal of such parathyroids does not bring about a cure of the hyperparathyroidism. In uremia, they found parathyroid hyperplasia of the diffuse type. This was attributed to the decrease of blood calcium, which in turn is caused by the retention of phosphates by the insufficient kidneys.

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7, **Dr. H. Edward MacMahon.** Dr. MacMahon demonstrated numerous mitoses in the cardiac muscle fibers of children under the age of two years. He offered evidence that there may be an increase in the number of cardiac muscle fibers after birth. The general impression of pathologists has been that the cardiac muscle fibers do not increase in number in postnatal life.

8, **Dr. Alexander Nedzel.** Dr. Nedzel produced lesions of the heart valves in dogs by injections of pitressin. In one group of animals, he used pitressin only and obtained an increase of cells and fibers in the heart valves. When bacteria (staphylococci or streptococci) were introduced intravenously in animals which had been injected with pitressin, he found that the organisms invaded the leaflets from their surfaces. The common opinion is that bacteria invade the valves from within, but Nedzel shows that the invasion apparently takes place from the surface. He was able to obtain a definite fibrosis of the valves and the proliferative type of inflammation from the use of pitressin alone.

9, **Dr. Sidney Farber.** Dr. Farber cut the vagus nerves in guinea pigs and rabbits. In both instances, the animals died of pulmonary edema, the guinea pigs in four or five hours, the rabbits in eight to thirty hours. A severe dyspnea develops and the alveoli fill with fluid. There is intense congestion of the veins and capillaries in the lungs and a variable amount of bronchopneumonia. The effect is something like a bulbar paralysis. He was able to exclude the larynx as a causative factor in the edema, since with artificial respiration through a tracheal cannula, the same pathologic picture developed. He believes that the most important factor in the edema is loss of vasomotor control.

10, **Dr. Valy Menkin.** Dr. Menkin reported further studies of the substance which he has isolated from areas of acute inflammation. This substance may be crystallized out. It is dializable and heat stable. He thinks it may be a polypeptid. When this substance is injected into normal areas of skin, it provokes increased capillary permeability.

11, **Dr. Milton C. Winternitz, Dr. R. M. Thomas,** and **Dr. P. M. LeCompte.** Dr. Winternitz reported an excellent study of the vasa vasorum. He showed numerous illustrations of the manner in which the blood vessels in the wall of an artery are distributed to its various layers. Evidence was offered which suggests that arteriosclerosis may be related to disturbances in the vascularity of the wall of the artery itself.

12, **Dr. Graham Ross, Dr. Theodore R. Waugh,** and **Dr. H. Tait Malloy.** The authors studied icterus neonatorum. They concluded that this is not a pure hemolytic change. They found no evidence in the blood of excessive hemopoietic activity. No complete explanation of the jaundice was found.

On the morning of March 26 a symposium on cancer was held by the Association of Pathologists and Bacteriologists.

13, **Dr. Arthur J. Vorwald** and **Dr. John W. Karr.** In a study of pneumoconiosis, the authors decided that there is no increased incidence in carcinoma of the lung in association with any form of pneumoconiosis. The carcinomas of the lung that occur in the Schneeberg miners develop on a basis of pneumoconiosis due to a radioactive dust.

14, **Dr. M. J. Shear.** Dr. Shear reported his studies on the carcinogenic activity of methyl derivatives of 1, 2-benzanthracene. He found that both cholanthrene and methyl-cholanthrene are highly carcinogenic and produce tumors within three or four months. For carcinogenic activity it is not necessary that the methyl group should be in position 6. He discussed the relation of chemical structure to carcinogenesis.

15, **Dr. W. C. Hueper** and **Dr. H. D. Wolfe.** Dr. Hueper produced aniline tumors in the bladders of dogs. Some of the papillomas developed into carcinomas. The chemicals were given by mouth.

16, **Dr. Balduin Lucké** Dr. Lucké has seen 500 examples of carcinoma in the frog's kidneys. The tumor always develops in the kidney and is structurally an adenocarcinoma. It shows large cell inclusions in the nuclei. Some of the tumors produce metastases. The tumor grows easily in vitro and the tumor cells outgrow the fibroblasts. No cell inclusions were seen in the cells of the cultures. Transplants do not seem to survive well except in the kidney, in which organ it is readily transplantable. This tumor occurs in frogs in the region of Chicago as well as in New England.

17, **Dr. Caspar G. Burn, Dr. Aline U. Orten,** and **Dr. Arthur H. Smith.** Dr. Burn and hi sassociates maintained rats for a long time on a diet markedly deficient in vitamin A. They developed xerophthalmia and cornification of the vagina. A massive enlargement of the jaws was obtained, which was due to the formation of large odontomas. It appeared that these growths were not neoplasms in the strict sense, but an irregular growth of the teeth.

18, **Dr. Robert A. Moore** and **Dr. Robert H. Melchionna.** Dr. Moore injected the prostate of animals with 5 per cent benzopyrene in lard. After four or five months large prostatic cancers developed. The cancers were for the most part squamous cell carcinomas, but a few sarcomas were obtained. Seventy per cent of the growths appeared after one hundred fifty days. Castration had no effect. The lard alone gives only a foreign body reaction. The squamous cells arise by metaplasia of the glandular epithelium. He thinks that the sarcomas arise from the muscle.

19, **Dr. Carl V. Weller.** Dr. Weller gave a general survey of the influence of heredity in the genesis of tumors. He showed that in a few types of tumors, notably retinoblastomas, the hereditary intrinsic factor is of great importance. The behavior of this particular tumor suggests that it is determined largely by intrinsic factors and that extrinsic factors are of little importance. He concluded that cancer depends for its development on both intrinsic and extrinsic factors and that the importance of these two influences varies in different types of tumors.

20, **Dr. S. Burt Wolbach.** Dr. Wolbach reported a detailed study of the histogenesis of experimental tar tumors. He illustrated the various stages in the development of the carcinomas from a stage of simple chronic inflammation to a definite carcinoma.

21, **Dr. Peyton Rous.** Dr. Rous gave a survey of the relation of filterable agents to the formation of tumors. His most interesting experiment was obtained as follows: The ears of a rabbit were tarred for about three months. This does not produce a neoplasm. In order to obtain carcinoma of the rabbit's ear, the tarring must be continued for a very long time. However, when the ears were tarred for about three months and the rabbit was then given an intravenous injection of the Shoppe papilloma virus, extensive squamous cell carcinoma developed in both ears. This experiment shows that the virus attacks cells which have been injured by tar and quickly leads to the formation of carcinoma.

22, **Dr. Leo Loeb.** Dr. Loeb gave an excellent survey of the relation of the sex hormones to tumors. Loeb did the pioneer investigations in this field and he surveyed the various lines of work which have been followed in recent years.

Book Reviews

The Physiology and Pharmacology of the Pituitary Body. By H. B. Van Dyke. Cloth. Pp. 577, with 55 illustrations. Chicago, 1936, The University of Chicago Press. $4.50.

Under the above title, the author, an active investigator in the field of pituitary physiology and pharmacology, has presented the most admirable monograph on the subject that has yet come to the reviewer's attention. Because of its completeness and up-to-dateness, the volume should be invaluable to clinicians as well as to teachers and research workers who are interested in the complex problems involving this important gland. In spite of the enormity of the task of sifting the essential data from several thousand original papers, the author has presented a comprehensive and critical résumé of the literature with amazing brevity and clearness. Well-established facts are catalogued in their proper sequence without unnecessary repetition. Controversial or incomplete data are presented fairly and in such a manner as to stimulate further investigation. The printing and illustrations are in keeping with the high quality of the text.

Because of the multiplicity of functions attributed to the pituitary body and the many interrelationships between it and other glands of internal secretion, a monograph on this subject must necessarily deal to some extent with the physiology and functional pathology of most of the other units of the endocrine system. Therefore, the general field of metabolism and such specialized but closely related fields as the physiology of reproduction are discussed with reference to their direct or indirect relationship to the pituitary. Clinical data are included, in so far as they contribute to the elucidation of the underlying pituitary physiology.

The first chapter of the book appropriately deals with the anatomy of the gland from the points of view of its embryology and comparative anatomy. Under this chapter such important features as the blood supply, lymph drainage, and nerve supply are detailed along with adequate descriptions of the histology of the pars glandularis, pars intermedia, pars tuberalis, and pars neuralis. The physiologic anatomy, as affected by such factors as pregnancy, castration, and hibernation, is clearly outlined. The probable sources of the known hormones from specific cells and their possible routes of transport from the gland are discussed.

The succeeding chapters deal with the physiologic, pharmacologic, and clinical aspects of the subject. Throughout the text, the confusing variations in functions of the gland at different levels in the animal scale are brought out by separate discussions for fish, amphibia, reptiles, birds, and mammals. The characteristic anatomic and physiologic effects of removal of the entire gland or of its separate parts are recorded for different animal species. Conversely, the effects on the pituitary body itself of removal of other glands, such as the thyroid, adrenals, pancreas and gonads, are reviewed. The rôle of the hypothalamus in functions attributed to the pituitary, particularly as regards the control of water, mineral, carbohydrate, and fat metabolism, is discussed fully, and gaps in our knowledge regarding this important question are pointed out. Special chapters are devoted to discussions regarding the growth-promoting, gonadotropic, lactogenic, and thyrotropic hormones and to interrelationships between the pars glandularis and the adrenals, pancreas,

parathyroids, and thymus. Other sections deal with evidence for considering the pars intermedia, pars tuberalis, and pars neuralis of the posterior lobe as true internal secretory structures. The melanophore-expanding substance of the pars intermedia can be extracted from the mammalian as well as from the amphibian pituitary, but the suggested rôle of such a "pigment hormone" in connection with the visual function in mammals has not as yet been established. Regarding the posterior lobe in mammals, the author concludes that, "extirpation experiments offer little support for the belief that the pars neuralis is physiologically important." Nevertheless, it is recognized that the two active substances which can be extracted from it; namely, the oxytocic and the pressor or diuretic-antidiuretic principles, exert striking pharmacologic effects and so may serve emergency functions in the intact animal.

A unique and helpful feature of the book is an appendix which gives a classified list of the scientific and commercial names of all of the known hormones and hormone preparations. The known structural formulas of the hormones are also given.

Of the more than five thousand papers reviewed by the author, three thousand are listed alphabetically in the bibliography under their complete titles. This constitutes one of the most valuable features of the monograph.

Operative Surgery. By J. Shelton Horsley and Isaac A. Bigger. Two vols. Ed. 4. Pp. 1,387, with 1,259 illustrations. St. Louis, 1937, The C. V. Mosby Co. $10.

Dr. Horsley, in revising material for the fourth edition of this text, was aware of the numerous difficulties besetting a single author in attempting to cover the entire scope of operative surgery adequately. He accordingly associated as co-authors surgeons of unquestionable repute who were not only trained in general surgery, but who had also devoted their efforts to various specialties. The co-authorship was allocated to Dr. Bigger, and the contributing authors were selected by these two men.

The fourth edition appears for the first time in two volumes, necessitated by the addition of new concepts of surgery and the inclusion of new material. The plan of the book duplicates that of its predecessors. All operative procedures presented are based upon established anatomic and physiologic principles, stressing that the aim of the surgeon should not be confined to meticulous and beautiful dissections of anatomic landmarks alone, but should also be directed toward the establishment of normally functioning organs postoperatively, as well as adequate removal of the pathology.

The indications, contraindications, advantages, and disadvantages for the various technical procedures are carefully outlined, followed by a discussion of the types of operations which, used by the authors, have produced the best results. In cases in which they believe that two or more procedures are adequate, they are likewise presented, the choice then being for the reader to decide as best suits his individual case. It is not a text of theoretic procedures, unproved and unaccepted, but instead a presentation of those procedures which the authors themselves performed and accepted as best for the restoration of the patient to a normal existence.

Each specialty of surgery is covered, and the newer procedures, such as those in the sympathetic nervous system and chest, are included. The reader may not agree with the choice of technique in some instances, and may believe that certain described methods could be replaced. Especially is this true in the chapter on transfusion, wherein the Lindemann method is emphasized and simpler methods omitted.

The bibliographies at the end of each chapter are short and well chosen, but in many references the page numbers are lacking. The illustrations, most of which are original and made especially for this book, are beautifully done. They are delicate and successful, combining a realistic quality with the diagrammatic.

As a reference book it is lacking in that only certain chosen surgical procedures are listed, and numerous others are omitted.

The book is best described as a monograph presenting the authors' choice and reasons for their personal selections.

Books Received

The receipt of books is acknowledged in this section and this statement must be regarded as sufficient acknowledgment of the courtesy of the sender. Selections will be made for more extensive review dictated by the interests of our readers and as space permits.

INHALATION ANESTHESIA: A FUNDAMENTAL GUIDE. By Arthur E. Goedel, Associate Clinical Professor of Surgery (Anesthesia), University of Southern California. Macmillan Surgical Monographs, Elliott C. Cutler, editor. Cloth. Price $2.50. Pp. 172. New York, 1937, The Macmillan Company.

PREOPERATIVE AND POSTOPERATIVE TREATMENT. By Robert L. Mason, Assistant in Surgery at the Massachusetts General Hospital. Cloth. Price $6.00. Pp. 495, with 123 illustrations. Philadelphia, 1937, W. B. Saunders Company.

ANLEITUNG ZUR SCHMERZBETÄUBUNG, Kurzes Lehrbuch der Lokalanästhesie, Allgemeinnarkose und sonstiger Anwendung der Betäubungsverfahren. By Fritz F. Härtel, Director of the Division of Surgery of Oskar-Ziethen-Krankenhauses Berlin-Lichtenberg; unter Mitwirkung von Dr. Horst Jencio. Pamphlet, price 10 M.; Bound, price, 11.50 M. Pp. 106, with 17 illustrations. Dresden, 1936, Theodor Steinkopff.

CHRISTIAN R. HOLMES: MAN AND PHYSICIAN. By Martin Fischer, University of Cincinnati. Cloth. Price $4. Springfield, Ill., 1937, Charles C. Thomas, Publisher.

DIABETES: A MODERN MANUAL. By Anthony M. Sindoni; introduction by Morris Fishbein; foreword by George Morris Piersol. Whittlesey House Health Series, Morris Fishbein, editor. Cloth. Price $2. Pp. 240. New York, 1937, Whittlesey House, McGraw-Hill Book Company.

A MANUAL OF RADIOLOGICAL DIAGNOSIS. For students and general practitioners. By Ivan C. C. Tchaperoff, Assistant Radiologist and Radium Registrar, St. Thomas' Hospital, London. With foreword by Philip H. Mitchiner. Cloth. Price $6. Pp. 256, with 286 illustrations. Baltimore, 1937, William Wood & Company.

DIAGNOSTIC ROENTGENOLOGY. Ross Golden, editor. Fabrikoid. Price $20. Pp. 854, with 964 illustrations. New York, 1936, Thomas Nelson & Sons.